PB88-251202

Health and Environmental Effects Profile for Ethylbenzene

(U.S.) Environmental Protection Agency
Cincinnati, OH

Jun 86

U.S. Department of Commerce
National Technical Information Service
NTIS

TECHNICAL REPORT DATA (Please read Instructions on the reverse before completing)		
1. REPORT NO. EPA/600/X-86/145	2.	3. RECIPIENT'S ACCESSION NO. PB88-251202
4. TITLE AND SUBTITLE Health and Environmental Effects Profile for Ethylbenzene		5. REPORT DATE June 1986
		6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S)		8. PERFORMING ORGANIZATION REPORT NO.
9. PERFORMING ORGANIZATION NAME AND ADDRESS		10. PROGRAM ELEMENT NO.
		11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS Environmental Criteria and Assessment Office Office of Research and Development U.S. Environmental Protection Agency Cincinnati, OH 45268		13. TYPE OF REPORT AND PERIOD COVERED
		14. SPONSORING AGENCY CODE EPA/600/22
15. SUPPLEMENTARY NOTES		

16. ABSTRACT

The Health and Environmental Effects Profile for Ethylbenzene was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Ethylbenzene has been determined to be a systemic toxicant. An Acceptable Daily Intake (ADI), defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect, for ethylbenzene is 0.097 mg/kg/day for oral exposure.

The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. The RQ value for ethylbenzene is 1000.

17. KEY WORDS AND DOCUMENT ANALYSIS		
a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field Group
18. DISTRIBUTION STATEMENT Public	19. SECURITY CLASS (This Report) Unclassified	21. NO. OF PAGES 120
	20. SECURITY CLASS (This page) Unclassified	22. PRICE A06 19.95

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

EPA/600/X-86/145
June 1986

PB88-251202

HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR ETHYLBENZENE

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
CINCINNATI, OH 45268

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

DISCLAIMER

This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3112. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

ACKNOWLEDGEMENTS

The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

Scientists from the following U.S. EPA offices provided review comments for this document series:

Environmental Criteria and Assessment Office, Cincinnati, OH
Carcinogen Assessment Group
Exposure Assessment Group
Reproductive Effects Assessment Group

Editorial review for the document series was provided by:
Judith Olsen and Erma Durden
Environmental Criteria and Assessment Office, Cincinnati, OH

Technical support services for this document series were provided by:
Bette Zwayer, Pat Daunt, Karen Mann, and Jacky Bohanon
Environmental Criteria and Assessment Office, Cincinnati, OH.

EXECUTIVE SUMMARY

Ethylbenzene is a colorless liquid with an aromatic odor at room temperature. It is soluble in many organic solvents, but is almost insoluble in water (Hawley, 1981). This chemical is manufactured by 12 U.S. chemical companies in 12 different sites. The total U.S. production volume of ethylbenzene in 1984 was 7562 million pounds (USITC, 1985). Ethylbenzene used for commercial purposes is derived primarily from petroleum and, to a lesser extent, from by-products of coke oven operations (NAS, 1980). Ethylbenzene is produced for chemical use by alkylation of benzene by the classic Friedel-Crafts reaction (Lewis et al., 1983). As a pure substance, >99% of ethylbenzene is used as a solvent or is exported (CMR, 1983).

The primary atmospheric process responsible for the removal of ethylbenzene is its reaction with OH• radicals. From the data available for the rate constant for this reaction (Ravishankara et al., 1978) and the concentration of OH• radicals in the troposphere (Cupitt, 1980), the average tropospheric half-life of ethylbenzene has been estimated to be >1 day. In polluted atmosphere, the half-life is expected to be shorter (Kopczynski et al., 1972; Heuss and Glasson, 1968; Dilling et al., 1976). Compounds with such relatively short half-lives are not expected to transport significantly from the troposphere to the stratosphere.

Although small amounts (4-9 ng/ℓ) of ethylbenzene have been detected in rainwater (Pankow et al., 1984; Kawamura and Kaplan, 1983), physical removal mechanisms, such as precipitation through rainfall or snowfall, through disposition of adsorbed aerosol particles and removal through impaction at the earth's surface, are not likely to remove significant amounts of

atmospheric ethylbenzene. A few investigators have demonstrated the possibility of aerosol transport of ethylbenzene from its source of emission to areas substantially further downwind (>100 miles) (Sexton and Westberg, 1980a,b).

The most significant loss process for aquatic ethylbenzene is volatilization. The half-life for volatilization is estimated to range between <1 day and a few days (Lyman et al., 1982; Mabey et al., 1981). In summer months, however, when the temperature of water is higher than in other seasons, microbial degradation may be significant and may be competitive with volatilization process (Gschwend et al., 1982). Significant removal of ethylbenzene through sorption onto particulate matters is not likely (Gschwend et al., 1982; Wakeham et al., 1983). Ethylbenzene will not significantly bioconcentrate in aquatic organisms (Nunes and Benville, 1979; Ogata et al., 1984).

Limited experimental data are available on the fate and transport of ethylbenzene in soil. Volatilization of ethylbenzene from the soil surface is likely to be a significant loss process (Bouwer et al., 1984). Biodegradation of ethylbenzene in aerobic soil is likely to be competitive with the volatilization process (Bouwer et al., 1984; Kappeler and Wuhrmann, 1978a,b; Battermann and Werner, 1984). From the measured concentrations of ethylbenzene at different soil depths at a waste site, Zoeteman et al. (1981) estimated the overall half-life of ethylbenzene in the disposal site to be ~37 days.

The median concentrations of ethylbenzene in rural/remote areas, urban/suburban areas and source areas in the United States during the period 1973-1980 were 470 ppt (2040 ng/m^3), 1200 ppt (5220 ng/m^3) and 630 ppt

(2740 ng/m³), respectively (Brodzinsky and Singh, 1982). The concentrations of ethylbenzene monitored in the United States during the 1980s remained fairly unchanged compared with the pre-1980 values given by Brodzinsky and Singh (1982). Localized concentrations of ethylbenzene in enclosed high traffic density areas, such as tunnels, could be >20 times higher than ambient levels (Kebbekus et al., 1983). The level of ethylbenzene in the occupational atmosphere may be as high as 41,000 ppb, as found in the histology laboratory (Angerer and Lehnert, 1979). Under certain conditions, the level of ethylbenzene in indoor air may be 2-3 times higher than outdoor air (Wallace et al., 1983; Hawthorne et al., 1983). Assuming that an individual inhales 20 m³ of air/day and that the concentration of ethylbenzene in rural/remote areas, urban/suburban areas and source areas is 2.04, 5.22 and 2.74 μg/m³, respectively (Brodzinsky and Singh, 1982), the daily inhalation exposure to U.S. populations residing in these corresponding areas are 41, 104 and 55 μg.

Ethylbenzene has been detected in natural surface water, industrial effluents, treated municipal effluents, leachates from contaminated sites and well waters. Staples et al. (1985) detected ethylbenzene in 7.5% of 1368 effluent samples at a median concentration of <3.0 μg/ℓ. The surface and groundwater samples showed ethylbenzene with a frequency of 10% and a median concentration of <5 μg/ℓ. The concentration of ethylbenzene in U.S. drinking water can vary from 0-40 μg/ℓ. On the basis of an average consumption of 2 ℓ of drinking water/day, the daily exposure to ethylbenzene from drinking water can vary from 0-80 μg; however, the vast majority of the U.S. population would be exposed to the lowest dosage of ethylbenzene from ingestion of drinking water.

Although ethylbenzene has been detected in a few food products (Mottram et al., 1984; U.S. EPA, 1980), the exposure to ethylbenzene from food ingestion by the U.S. population cannot be estimated unless its levels in composite food samples are known.

Limited information concerning toxicity of ethylbenzene to aquatic organisms is available. Acutely toxic concentrations range from 0.42 mg/ℓ for the bay shrimp (Benville and Korn, 1977) to 1030 mg/ℓ for the Pacific oyster (LeGore, 1974). The most sensitive plant species tested was the blue-green algae, Microcystis aeruginosa, with a toxicity threshold of 33 mg/ℓ (Bringmann and Kuehn, 1978), while the bacterium, Pseudemonas putida, had a toxicity threshold of 12 mg/ℓ (Bringmann and Kuehn, 1976).

At least 82% of the administered dose of ethylbenzene was absorbed from the gastrointestinal tract after oral exposure in rats (Climie et al., 1983), whereas 44 and 64% of the administered dose of ethylbenzene was absorbed after inhalation exposure of rats (Chin et al., 1980) and humans (Bardodej and Bardodejova, 1970), respectively. Of the administered radioactivity, <0.2% was recovered from the organs and carcasses of rats within 48-72 hours after inhalation exposure (Chin et al., 1980), indicating that substantial amounts of ethylbenzene are not retained. Furthermore, ethylbenzene did not accumulate with time in the perirenal fat of rats exposed to 50-600 ppm (218-2610 mg/m^3) of ethylbenzene for 6 hours/day, 5 days/week for up to 16 weeks (Engstrom et al., 1985). Approximately 82% of the administered radioactivity was recovered from the urine of rats treated with a single oral dose of ethylbenzene; 83% of the absorbed radioactivity was recovered from the urine of rats (Chin et al., 1980; Engstrom, 1984) and 94% was recovered from the urine of humans (Bardodej and Bardodejova, 1970) exposed to ethylbenzene by inhalation. In rats, only 1.5% of the orally administered dose (Climie et al., 1983) and 0.7% of the absorbed inhalation

dose (Chin et al., 1980) was excreted in the feces. Excretion by the lungs of the absorbed inhalation dose was ~8% in rats (Chin et al., 1980). Mandelic (23%) and hippuric acid (34%) accounted for more than half of the radioactivity recovered from the urine of rats treated with a single oral dose of ethylbenzene (Climie et al., 1983). Mandelic acid (23-28%), phenylethanol (23-25%) and benzoic acid (24-27%), however, accounted for ~75% of the radioactivity recovered from the urine of the rats after a single 6-hour inhalation exposure to ethylbenzene (Engstrom, 1984), indicating that the metabolic pathways are sensitive to the route of administration. With subchronic inhalation exposure to ethylbenzene, the ratio of the three major urinary metabolites in rats (mandelic, hippuric and benzoic acids) and phenylethanol was more sensitive to dose than to duration, and did not appear to change over time, with the possible exception of mandelic acid which accounted for a statistically significant decreased ($p<0.05$) percentage of the total urinary metabolites after 9 weeks of treatment (Engstrom et al., 1985). After a single 8-hour inhalation exposure of humans to ethylbenzene, 64% of the retained dose was identified in the urine as mandelic acid and 25% as phenylglyoxylic acid (Bardodej and Bardodejova, 1970), indicating that there are species differences in the metabolism of ethylbenzene even when administered by the same route. There are species differences in the metabolism of ethylbenzene after oral administration as well. Mandelic (23%) and hippuric (34%) acids were the major urinary metabolites after a single oral dose of ethylbenzene was administered to rats (Climie et al., 1983); however, methylphenylcarbinyl glucosiduronic (30-35%) and hippuric (30-35%) acids were the major urinary metabolites after a single oral dose of ethylbenzene was administered to rabbits (El Masry et al., 1956). Mandelic acid accounted for only 2% of the dose administered to rabbits.

Pertinent data regarding the carcinogenicity of ethylbenzene alone were not located in the available literature cited in the Appendix. Studies of hazards to workers employed in the production of styrene products, which may involve exposure to ethylbenzene, are confounded by concurrent exposure to such known carcinogens as benzene, butadiene, acrylonitrile and formaldehyde (NCI, 1985). Ethylbenzene was not mutagenic in bacteria (Florin et al., 1980; Nestmann et al., 1980; Douglas et al., 1980), yeast (Nestman and Lee, 1983) or Drosophila melanogaster (Donner et al., 1980), but was weakly mutagenic to human lymphocytes in vitro, as evaluated by the SCE assay (Norppa and Vainio, 1983). Ethylbenzene had no significant adverse effect on fetal development and was not teratogenic to rabbits at inhalation concentrations ≤1000 ppm (≤4350 mg/m³), 7 hours/day during gestation (Hardin et al., 1981; Andrew et al., 1981). Rat fetuses had increased incidences of extra ribs following exposure of dams to 96 or 959 ppm (418 or 4172 mg/m³), 7 hours/day during gestation. Increased liver and kidney weights accompanied by hepatic and renal histopathology (cloudy swelling) were observed in rats treated orally with ≥408 mg/kg/day of ethylbenzene, 5 days/week for 6 months (Wolf et al., 1956). After inhalation exposure for 6 months, similar hepatic and renal effects were observed in rats exposed to ≥5400 mg/m³, 7 hours/day, 5 days/week (Wolf et al., 1956). Increased liver and kidney weights also occurred at ≥1700 mg/m³, 7 hours/day, 5 days/week. After 6 months of inhalation exposure, guinea pigs had slightly decreased growth rates at 5400 mg/m³ and slightly increased liver weights at 2600 mg/m³, 7 hours/day, 5 days/week (Wolf et al., 1956). Testicular changes characterized by germinal epithelial degeneration were observed in one rabbit and one monkey at 2600 mg/m³, 7 hours/day, 5 days/week (Wolf et al., 1956).

The monkeys also had slightly increased liver weights. Hepatic and renal enzyme levels were increased in rats exposed by inhalation to 300 or 600 ppm (1305 or 2610 mg/m^3) ethylbenzene, 6 hours/day, 5 days/week for up to 16 weeks (Elovaara et al., 1984).

The ADI for ethylbenzene is 0.097 mg/kg/day or 6.8 mg/day for a 70 kg man, based on a NOEL of 97 mg/kg/day in rats treated orally for ~6 months (Wolf et al., 1956) and using an uncertainty factor of 1000. Higher doses ($\geq$291 mg/kg/day) resulted in increased kidney and liver weights accompanied by cloudy swelling in these organs; these data were used as the basis of the RQ of 1000. Ethylbenzene is an IARC Group 3 chemical; that is, it cannot be classified as to its carcinogenicity in humans. The analogous U.S. EPA, CAG classification is Group D.

TABLE OF CONTENTS

		Page
1.	INTRODUCTION	1
1.1.	STRUCTURE AND CAS NUMBER	1
1.2.	PHYSICAL AND CHEMICAL PROPERTIES	1
1.3.	PRODUCTION DATA	3
1.4.	USE DATA	3
1.5.	SUMMARY	5
2.	ENVIRONMENTAL FATE AND TRANSPORT PROCESSES	6
2.1.	AIR	6
2.2.	WATER	11
2.3.	SOIL	14
2.4.	SUMMARY	16
3.	EXPOSURE	18
3.1.	AIR	18
3.2.	WATER	23
3.3.	FOOD	26
3.4.	SUMMARY	26
4.	PHARMACOKINETCS	28
4.1.	ABSORPTION	28
4.2.	DISTRIBUTION	28
4.3.	METABOLISM	29
4.4.	EXCRETION	32
4.5.	SUMMARY	37
5.	EFFECTS	41
5.1.	CARCINOGENICITY	41
5.2.	MUTAGENICITY	41
5.3.	TERATOGENICITY	43
5.4.	OTHER REPRODUCTIVE EFFECTS	46
5.5.	CHRONIC AND SUBCHRONIC TOXICITY	46
5.6.	OTHER RELEVANT INFORMATION	49
5.7.	SUMMARY	50
6.	AQUATIC TOXICITY	52
6.1.	ACUTE	52
6.2.	CHRONIC	52
6.3.	PLANTS	52
6.4.	RESIDUES	56
6.5.	SUMMARY	56

TABLE OF CONTENTS (cont.)

	Page
7. EXISTING GUIDELINES AND STANDARDS	57
7.1. HUMAN	57
7.2. AQUATIC	57
8. RISK ASSESSMENT	58
9. REPORTABLE QUANTITIES	60
9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY	60
9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY	66
10. REFERENCES	68
APPENDIX: LITERATURE SEARCHED	101

LIST OF TABLES

No.	Title	Page
1-1	U.S. Producers of Ethylbenzene and Their Annual Capacities. .	4
2-1	Relative Reactivities of Benzene and Ethylbenzene based on NO_2 Formation Rate.	9
3-1	Concentration of Ethylbenzene in a Few U.S. and Foreign Locations in the 1980s.	19
3-2	Concentrations of Ethylbenzene in Occupational Situations . .	22
4-1	Concentration of Ethylbenzene in the Perirenal Fat of Rats After 2, 5, 9 and 16 Weeks of Exposure.	30
4-2	Percentage Composition of the Urinary Metabolite Pattern of Ethylbenzene at Two Exposure Levels	34
4-3	Recovery of Radioactivity During 24 Hours After Exposure of Rats to 1000 mg/m³ of ^{14}C-Ethylbenzene for 6 hours.	36
4-4	Urinary Excretion of Ethylbenzene	38
5-1	Mutagenicity Testing of Ethylbenzene.	42
5-2	Percent of Mated Female Rats That Were Pregnant After Exposure to Ethylbenzene.	45
5-3	Summary of Ethylbenzene Exposure Levels, Duration of Exposure and Results in Rats, Guinea Pigs, Rabbits and Monkeys	48
6-1	Acute Toxicity of Ethylbenzene to Fishes.	53
6-2	Acute Toxicity of Ethylbenzene to Invertebrates	55
9-1	Toxicity Summary for Ethylbenzene	61
9-2	Composite Scores for Ethylbenzene	63
9-3	Ethylbenzene: Minimum Effective Dose (MED) and Reportable Quantity (RQ)	65

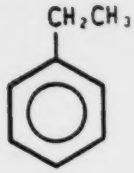
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
BUN	Blood urea nitrogen
CAS	Chemical Abstract Service
CNS	Central nervous system
EC_{50}	Concentration effective to 50% of recipients
K_{oc}	Soil sorption coefficient
K_{ow}	Octanol/water partition coefficient
LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
NADPH	Nicotinamide adenine dinucleotide phosphate
NOAEL	No-observed-adverse-effect level
NOEC	No-observed-effect concentration
NOEL	No-observed-effect level
ppb	Parts per billion
ppm	Parts per million
ppt	Parts per thousand
RQ	Reportable quantity
SCE	Sister chromatid exchange
SD	Standard deviation
SGPT	Serum glutamic pyruvic transaminase
STEL	Short-term exposed level
TLV	Threshold limit value
UV	Ultraviolet

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

The chemical commonly called ethylbenzene is also known by the synonyms ethylbenzol and phenylethane. The structure, empirical formula, molecular weight and CAS Registry number for ethylbenzene are as follows:



Empirical formula: C_8H_{10}

Molecular weight: 106.17

CAS Registry number: 100-41-4

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Ethylbenzene is a colorless liquid with an aromatic odor at room temperature. It is soluble in ethyl alcohol, benzene, carbon tetrachloride and ethyl ether, but is almost insoluble in water (Hawley, 1981). The physical properties of ethylbenzene are listed below:

Melting point:	-94.97°C	Verschueren, 1983
Boiling point: (1 atm)	136.2°C	Verschueren, 1983
Specific gravity: at 20/4°C	0.867	Verschueren, 1983
Flash point:	15°C	Hawley, 1981
Autoignition temperature:	432°C	Hawley, 1981
Viscosity: at 25°C	0.64 (centipoise)	Hawley, 1981
Vapor pressure (mm Hg):		
at 20°C	7	Verschueren, 1983
at 25°C	9.5	Mackay and Shiu, 1981
at 27°C	10.4	Chao et al., 1983
at 30°C	12	Verschueren, 1983

Water solubility: at 25°C	161 mg/ℓ	NAS, 1980
Henry's Law Constant at 25°C	8.24×10^{-3} atm•m³/mol (estimated from vapor pressure and solubility) 8.43×10^{-3} atm•m³/mol (experimental)	Mackay and Shiu, 1981
Log K_{ow}:	3.15	Hansch and Leo, 1985
Log K_{oc}:	2.22 2.61	Chiou et al., 1983 Sabljic, 1984
Air odor threshold:	2.3 ppm 32.2 ppm	Amoore and Hautala, 1983 Rosen et al., 1963
Conversion factor: at 20°C	1 mg/m³ = 0.23 ppm	

The solubility value for ethylbenzene in water at 25°C has been reported to vary between 152 and 208 mg/ℓ (McAuliffe, 1963, 1966; Sanemasa et al., 1981; Sutton and Calder, 1975; Brown and Wasik, 1974; Wasik et al., 1983; Bohon and Claussen, 1951; Schwarz and Miller, 1980; Tewari et al., 1982; Polak and Lu, 1973). Consequently, the estimated value for H has been reported to vary between 8.75×10^{-3} and 6.39×10^{-3} atm•m³/mol (Lyman et al., 1982; Mackay and Shiu, 1981; Shen, 1982)

Chemically, ethylbenzene can be oxidized to form hydroperoxide at high temperatures (Schoenberg et al., 1982). It can also be sulfonated (Knaggs et al., 1983). The dehydrogenation of ethylbenzene is used commercially for the production of styrene (Section 1.3.). None of these reactions, however, are environmentally significant. The environmental reactions of ethylbenzene are discussed in Chapter 2.

1.3. PRODUCTION DATA

The U.S. EPA's TSCA Production File (U.S. EPA, 1977) lists 22 U.S. companies manufacturing this chemical in 29 locations in 1977. The total U.S. production of ethylbenzene was 8312 million pounds in 1977 (USITC, 1978). Producers of ethylbenzene in the U.S. and their annual production capacities as of January 1, 1985 are listed in Table 1-1.

The USITC (1985) lists these same companies as the U.S. producers of ethylbenzene in 1984, as well as Cosden Oil and Chemical Co., Beaumont, TX, and Koppers Co., Inc., Follansbee, WV. The U.S. production volume of this chemical in 1984 was 7562 million pounds (USITC, 1985).

Ethylbenzene used for commercial purposes is derived primarily from petroleum and, to a lesser extent, from by-products of coke oven operations. The catalytic reforming process typically yields ~4% ethylbenzene in the reformate (NAS, 1980). Ethylbenzene is produced for chemical use by alkylation of benzene. Benzene is reacted with ethylene in the presence of a catalyst system to produce ethylbenzene by the classic Friedel-Crafts reaction. The crude ethylbenzene formed by this method is purified by a series of distillation steps. Several variations of this process are now used to produce commercial ethylbenzene (Lewis et al., 1983, MacFarlane, 1976; Dwyer et al., 1976).

1.4. USE DATA

Ethylbenzene is a constituent of gasoline. A composite gasoline sample obtained in Los Angeles contained 0.8 ethylbenzene (1.77% by weight) (NAS, 1980). As a pure substance, >99% of the ethylbenzene produced in the United States is used as an intermediate from styrene production; the remainder is used as a solvent or is exported (CMR, 1983).

TABLE 1-1

U.S. Producers of Ethylbenzene and Their Annual Capacities*

Company	Location	Annual Capacity (millions of pounds)
American Hoechst Corp.	Bayport, TX	1035
Arco	Channelview, TX	1160
Gulf Oil Corp.	St. James, LA	679
Cos-Mar, Inc.	Carville, LA	1730
Dow Chemical U.S.A.	Freeport, TX	1850
DuPont Co., Inc.	Chocolate Bayou, TX	60
El Paso Products Co.	Odessa, TX	330
Koch Industry, Inc.	Corpus Christi, TX	22
Monsanto Co.	Texas City, TX	1560
Standard Oil Co. (Indiana)	Texas City, TX	908
Total		9334

*Source: SRI, 1985

1.5. SUMMARY

Ethylbenzene is a colorless liquid with an aromatic odor at room temperature. It is soluble in many organic solvents, but is almost insoluble in water (Hawley, 1981). This chemical is manufactured by 12 U.S. chemical companies in 12 different sites. The total U.S. production volume of ethylbenzene in 1984 was 7562 million pounds (USITC, 1985). Ethylbenzene used for commercial purposes is derived primarily from petroleum and, to a lesser extent, from by-products of coke oven operations (NAS, 1980). Ethylbenzene is produced for chemical use by alkylation of benzene by the classic Friedel-Crafts reaction (Lewis et al., 1983). As a pure substance, >99% of ethylbenzene is used as a solvent or is exported (CMR, 1983).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. AIR

Ethylbenzene is ubiquitous in the atmosphere due to its widespread use. Plastic and rubber industries that use ethylbenzene in the manufacture of styrene can be sources of atmospheric ethylbenzene. Evaporation of gasoline that normally contains ethylbenzene also contributes to atmospheric ethylbenzene. Other sources of ethylbenzene in the atmosphere are solvents containing ethylbenzene, vehicular exhaust, jet engine exhaust, waste incineration and tobacco smoke (Harkov et al., 1983; Hampton et al., 1982; Esposito, 1968; Stavinoha and Newman, 1972; Higgins et al., 1983; Katzman and Libby, 1975; Junk and Ford, 1980; Holzer et al., 1976; Harkov, 1982; Black and High, 1977; Ball et al., 1970; Cook, 1980). Ethylbenzene also forms during forest fires (Westberg et al., 1981).

The primary process for the removal of atmospheric ethylbenzene is its reaction with OH• radicals. The rate constant for this reaction was determined as 7.95×10^{-12} cm^3 $molecule^{-1}$ sec^{-1} at 25°C (Ravishankara et al., 1978) and as 7.97×10^{-12} cm^3 $molecule^{-1}$ sec^{-1} at 32°C (Lloyd et al., 1976). If the concentration of OH• radicals in the troposphere is assumed to be 10^6 molecule cm^{-3} (Cupitt, 1980), the half-life of ethylbenzene in the troposphere at 25°C would be ~1 day; however, the average tropospheric temperature is -8°C (Dilling, 1982), and since the rate constant for the OH• radical reaction with ethylbenzene at -8°C is not available, it is not possible to estimate the average tropospheric half-life of ethylbenzene, but it is expected to be >1 day. Compounds with tropospheric half-lives of 1-2 days are not expected to transport significantly from the troposphere to the stratosphere.

The reactivity of ethylbenzene in the atmosphere can also be estimated by measuring the decrease of its concentration under simulated tropospheric conditions. Several investigators reported the results of irradiation of ethylbenzene in the presence of NO_x with artificial sunlight and natural sunlight sources. When irradiated with light of wavelength >290 nm, NO, NO_2 and O_3 will eventually produce OH• radicals in the presence of water vapor (Dilling, 1982). Therefore, the active species in these systems are OH• radicals. At an ethylbenzene to HO_x ratio of 2:1, Yanagihara et al. (1977) reported 32.5% disappearance of ethylbenzene in 5 hours. The disappearance rate is also reported as 19% in 6 hours (Kopczynski et al., 1972), 29% in 6 hours (Heuss and Glasson, 1968), 50% in 5 hours (Dilling et al., 1976) and 4% in 1 hour (Kopczynski, 1964). The difference in reaction rates is due to differences in initial concentrations of the reactants, in the nature of irradiation sources and in the nature of reaction vessels. In order to derive the half-life of ethylbenzene under atmospheric conditions from the smog chamber experiments, the ethylbenzene-to-NO_x ratio should be designed to simulate atmospheric conditions, the spectral distribution and intensity of the irradiation source should follow natural sunlight, the system should be simulated to contain other competing species that are present in natural atmosphere, and the reaction should be free from competing wall reactions. Since it is difficult to reproduce all these conditions, the half-life of ethylbenzene resulting from atmospheric reactions is not usually estimated from the smog chamber experiments. Instead, the smog chamber experiments are used to evaluate the comparative reactivities of organic solvents in the formation of photochemical smog. Since the reactions of the hydrocarbons with NO_x produce NO_2, O_3 and formaldehyde, the relative reactivities can be expressed in terms of the rate of formation

of these products (Levy, 1973; Farley, 1977). It has been shown by Dimitriades et al. (1975) that the relative reactivity based on rate of NO_2 formation agrees well with the experimental results of various investigators. The relative reactivity of ethylbenzene with respect to benzene as determined by various investigators based on the NO_2 formation rate is given in Table 2-1.

The photochemical reaction of ethylbenzene in the presence of NO_x yields ethylphenol, benzaldehyde, acetophenone, m- and p-nitrobenzene (NAS, 1980), in addition to O_3, NO_2 and formaldehyde. Peroxyacetyl nitrate was not detected as a reaction product (Heuss and Glasson, 1968; Yanagihara et al., 1977).

Other reactions, such as direct photolysis or reaction with O_3, peroxy radicals and atomic oxygen may remove some ethylbenzene from the atmosphere. The absorption spectra of ethylbenzene solution in methanol in the UV-visible range has been given by Sadtler (1966). The longest wavelength absorption maximum for ethylbenzene is 269 nm, and it does not significantly absorb light at wavelengths >280 nm (Sadtler, 1966). Therefore, direct photolysis of ethylbenzene in the atmosphere does not appear to be a significant fate process. The rate constant for ethylbenzene reaction with atomic oxygen (3P) is 5.3×10^{-12} cm^3 $molecule^{-1}$ sec^{-1} at 25°C (Herron and Huie, 1973).

If the concentration of atomic oxygen in the atmosphere is assumed to be 2.5×10^4 molecules cm^{-3} (Graedel, 1978), the estimated half-life of ethylbenzene in the atmosphere resulting from its reaction with atomic oxygen would be ~60 days. Therefore, this reaction is insignificant compared with its reaction with OH• radicals in the atmosphere. The estimated rate constants for the reaction of toluene with O^3 and peroxy

TABLE 2-1

Relative Reactivities of Benzene and Ethylbenzene
Based on NO_2 Formation Rate

Benzene	Ethylbenzene	Reference
1	3.6	Glass and Tuesday, 1970
1	2	Kopczynski, 1964
1	4.5	Heuss and Glasson, 1968
1	2.8	Yanagihara et al., 1977
1	4.0	Laity et al., 1973
1	3.0	Dimitriades et al., 1975

radicals have been given by NAS (1980). According to NAS (1980), these rates are applicable to higher alkylbenzenes, presumably including ethylbenzene; however, the rate of ethylbenzene removal from the atmosphere by O^3 and peroxy radical reaction is $5x10^{-5}$ and $4x10^{-8}$ times, respectively, less than its reaction with OH• radicals. The insignificance of O^3 and peroxy radical reactions with ethylbenzene compared with its reaction with OH• radicals in the removal of atmospheric ethylbenzene has been also shown by Hendry and Kenley (1979).

The three physical mechanisms responsible for the removal of a chemical from the atmosphere -- removal through dissolution in rainfall or snowfall, removal through adsorption on aerosol particles and subsequent deposition, and deposition through sorption on impaction with soil and water at the earth's surface -- are not likely to remove significant amounts of atmospheric ethylbenzene. This prediction is based on the study of Cupitt (1980), who showed that it is not possible to remove significant amounts of structurally similar compounds (toluene and xylenes) from the atmosphere through physical removal mechanisms. The presence of small amounts of ethylbenzene (4-9 ng/ℓ) in rainwater in Los Angeles, CA (Kawamura and Kaplan, 1983) and in Beaverton, OR (Pankow et al., 1984), however, demonstrates the possibility of removal of atmospheric ethylbenzene through rainwater.

The overall atmospheric half-life for ethylbenzene is ~1 day. Therefore, this compound is expected to be transported from the areas of emission to points substantially further downwind. For example, ethylbenzene emitted from two automobile plants in Sweden had its concentration reduced to one-third of its original value at a distance of 1 km downwind. The concentration at this point was still ~100 times higher than the background level

(Petersson, 1982). The long distance (up to >100 miles) transport of ethylbenzene in the air has been demonstrated by other authors (Sexton and Westberg, 1980a,b).

2.2. WATER

The oxidation rate constants for ethylbenzene reactions with singlet oxygen ($^1O_2$), peroxy radicals ($RO_2\cdot$) and alkoxy radicals ($RO\cdot$) are <360 M^{-1} $hour^{-1}$ (Mabey et al., 1981), 720 M^{-1} $hour^{-1}$ (Mabey et al., 1981) and 4.3×10^{-8} M^{-1} $hour^{-1}$, respectively (Hendry et al., 1974). If the concentrations of $^1O_2$, $RO_2\cdot$ and $RO\cdot$ are assumed to be 10^{-12}, 10^{-14} and 10^{-9} M, respectively, in water (Mill et al., 1982), the estimated half-lives for these reactions will be too high. Therefore, none of these processes will have significant effect on the fate of ethylbenzene in waterbodies.

The significant removal of ethylbenzene from water by hydrolysis or direct photolysis is not likely, since the hydrolysis rate constant for the compound has been reported to be 0 (Mabey et al., 1981), and the compound does not absorb light at wavelengths >290 nm; however, sensitized photoreaction of ethylbenzene in water may be possible. Ehrhardt and Petrick (1984) reported that ethylbenzene undergoes sensitized photoreaction in seawater with anthroquinone. Ethylbenzene photosensitized by humic substances present in natural bodies of water may undergo similar reactions; however, lack of rate data for this reaction precludes estimation of the half-life of ethylbenzene.

The biodegradability of ethylbenzene with both pure and mixed cultures of microorganisms has been studied by several investigators. Several species of pure cultures of microorganisms including Pseudomonas putida (Gibson et al., 1968 1973; Gibson and Yeh, 1973; Kanemitsu et al., 1980;

Gibson, 1977, 1978; Ribbons and Eaton, 1982), Nocardia sp. (Perry, 1979; Cox and Goldsmith, 1979; Jamison et al., 1970; Horvath, 1972) and Methylomonas sp. (Higgins et al., 1980, 1981) have been shown to biodegrade ethylbenzene. Similarly, mixed microorganisms from activated sludge and sewage seed have been shown to biodegrade ethylbenzene. It has been reported that the biodegradation of ethylbenzene requires acclimatization of the microorganisms (Thom and Agg, 1975). Thus, acclimatized activated sludge (Malaney and McKinney, 1966; Malaney, 1960; Feiler et al., 1979; Bogan and Sawyer, 1955; Marion and Malaney, 1964) and sewage seed (Bogan and Sawyer, 1955; Ludzack and Ettinger, 1963) have been reported to moderately biodegrade ethylbenzene. In a static-culture flask-screening procedure with domestic wastewater as microbial inoculum, ethylbenzene was reported to undergo significant degradation with rapid adaptation at an initial substrate concentration of 5 mg/ℓ. At this concentration, 100% of ethylbenzene underwent biodegradation in 7 days (Tabak et al., 1981). Activated sludge treatment of municipal wastewater in a treatment plant, at an initial ethylbenzene concentration of 148 µg/ℓ, was reported to remove 99% of ethylbenzene. Activated sludge treatment of industrial wastewater in a treatment facility, at an initial ethylbenzene concentration of 882 µg/ℓ, removed 83% of the compound. Similarly, aerated lagoon and PACT treatment of industrial wastewater was reported to remove $\geq$78% of the ethylbenzene from the wastewater (Patterson and Kodukala, 1981).

The rate of biodegradation of ethylbenzene in natural water has also been studied. Almost complete biodegradation of ethylbenzene occurred in 10 days when 1% gas oil containing 38 µℓ/ℓ of ethylbenzene was incubated with North Sea coast water at 25°C (Van Der Linden, 1978). From their monitoring experiments at a coastal site, Gschwend et al. (1982) concluded

that biodegradation may be moderately important for ethylbenzene, but under most favorable conditions, ethylbenzene can be degraded in a few days. The estimated biotransformation rate constant for ethylbenzene is 3×10^{-9} mℓ cell^{-1} hour^{-1} (Mabey et al., 1981). If the concentration of microorganisms capable of biodegrading ethylbenzene is assumed to be 5×10^{6} cell mℓ$^{-1}$ (Banergee et al., 1984; Oduntan and Odeyemi, 1984), the biodegradation half-life of ethylbenzene under most favorable conditions (high temperature and high microorganism concentration) will be ~2 days.

Volatilization appears to be the major route for the removal of this compound from aquatic environments. Using a wind velocity of 3 m/sec and water current of 1 m/sec, the mass transfer coefficient for ethylbenzene was estimated to be 22.3 cm/hour (Lyman et al., 1982). For a water depth of 1 m, the estimated half-life for evaporation is 3.1 hours. The evaporative half-life can also be estimated from the reaeration rate ratio. Based on a reaeration rate ratio of 0.489 (Mabey et al., 1981) and the oxygen reaeration rates in pond, river and lake water at 0.19, 0.96 and 0.24 day^{-1} (Mabey et al., 1981), respectively, the corresponding half-lives are 7.5, 1.5 and 5.9 days. Thus, the evaporative half-life of ethylbenzene in natural bodies of water is expected to range between <1 day and a few days. Volatilization as a major loss process for aquatic ethylbenzene was also confirmed by Gschwend et al. (1982), from their experimental measurement of ethylbenzene concentration in coastal water for a period of 15 months and the controlled ecosystem study of Wakeham et al. (1983).

The loss of ethylbenzene from the aquatic phase through sorption onto particulate matters can be predicted from the K_{oc} values. The log K_{oc} value of 2.22-2.61 (see Section 2.1.) for ethylbenzene indicates that sorption may not be an important process. Gschwend et al. (1982) studied the

absorptive loss of ethylbenzene onto sediment and concluded that adsorptive losses are small and may amount to only a few percent of total ethylbenzene in the aquatic phase. From their controlled ecosystem study, Wakeham et al. (1983) also concluded that adsorption to sediments may not be a significant removal mechanism for aquatic ethylbenzene.

The steady-state BCF of a chemical in aquatic organisms can be estimated from the equation: $\log BCF = 0.85 \log K_{ow} - 0.70$ (Lyman et al., 1982). Based on a $\log K_{ow}$ value of 3.15 (see Section 1.2.), the steady-state log BCF for ethylbenzene is estimated to be 1.98. The concentrations of ethylbenzene in the aqueous phase and tissue of the Manila clam, Tapes semidecussata, exposed to the same aqueous phase for a period of 8 days in a flow system was measured by Nunes and Benville (1979). From the experimental mean aquatic and tissue concentration, the log BCF value is estimated to be 0.6. The log BCF value for ethylbenzene in whole goldfish, Carassius auratus, was experimentally determined to be 1.19 (Ogata et al., 1984). Therefore, it is concluded that ethylbenzene will not significantly bioconcentrate in aquatic organisms.

2.3. SOIL

Limited experimental data are available on the fate and transport of ethylbenzene in soil. From the expected fate of this compound in aquatic media (see Section 2.2.), it is speculated that neither chemical reactions nor photochemical reactions will play significant roles in determining the fate of this compound in soil. The possibility of biodegradation of ethylbenzene in soil was studied by several investigators. A species of Nocardia isolated from soil was reported to oxidize ethylbenzene to phenylacetic acid (Davis and Raymond, 1961). The biodegradation of ethylbenzene with mixed microorganisms from soil was reported by Kappeler and Wuhrmann (1978a).

When an aqueous solution of gas oil containing ethylbenzene was percolated through a hydraulically unsaturated soil column, ethylbenzene underwent 100% degradation in 8 days after a 3- to 4-day lag period. Incubation experiments with natural microbial flora of groundwater degraded ethylbenzene completely in 12 days. Experiments with pure strains, Pseudomonas sp., isolated from the soil provided evidence of formation of 1-phenylethanol from ethylbenzene (Kappeler and Wuhrmann, 1978b). In situ biodegradation studies, where contaminated water was percolated through a large-scale field, reportedly reduced ethylbenzene concentration by 93% in 17 days (Battermann and Werner, 1984). Rapid infiltration of secondary wastewater containing ethylbenzene has been reported to reduce the concentration to >50% during soil percolation for a period of 3-7 days (Bouwer et al., 1984). Although the reduction in concentration may be due to biodegradation, volatilization and siphon processes, the last two mechanisms have been ruled out as significant removal processes (Bouwer et al., 1984). Bouwer and McCarty (1984) reported that the degradation to ethylbenzene occurs in soil under aerobic conditions, but significant degradation does not occur in soil under anaerobic conditions.

Data pertaining to the loss of ethylbenzene from soil through sorption and volatilization are limited. From the log K_{oc} value of 2.22-2.61 (see Section 1.2.), it can be predicted that ethylbenzene will not be strongly adsorbed onto soil. The experimental data of Kanatharano and Grob (1983) confirms this prediction. The sorption of ethylbenzene on three different soils was reported to be <5% by these investigators. Based on its volatilization capability from aquatic media (see Section 2.2.), it is predicted that volatilization will be a significant loss process from soil surfaces.

The infiltration study of Bouwer et al. (1984) showed that volatilization caused significant (35-53%) losses of xylenes and C_3-benzene isomers from soil surface. Since ethylbenzene is structurally similar to these compounds, surface volatilization from soil is likely to be a significant fate process for ethylbenzene. Finally, from the measured concentrations of ethylbenzene at different soil depths of a waste site at Noordwijk, The Netherlands, Zoeteman et al. (1981) estimated the overall half-life of ethylbenzene in the disposal site to be ~37 days.

2.4. SUMMARY

The primary atmospheric process responsible for the removal of ethylbenzene is its reaction with OH• radicals. From the data available for the rate constant for this reaction (Ravishankara et al., 1978) and the concentration of OH• radicals in the troposphere (Cupitt, 1980), the average tropospheric half-life of ethylbenzene has been estimated to be >1 day. In polluted atmosphere, the half-life is expected to be shorter (Kopczynsky et al., 1972; Heuss and Glasson, 1968; Dilling et al., 1976). Compounds with such relatively short half-lives are not expected to transport significantly from the troposphere to the stratosphere.

Although small amounts (4-9 ng/ℓ) of ethylbenzene have been detected in rainwater (Pankow et al., 1984; Kawamura and Kaplan, 1983), physical removal mechanisms, such as precipitation through rainfall or snowfall, deposition of adsorbed aerosol particles and removal through impaction at the earth's surface, are not likely to remove significant amounts of atmospheric ethylbenzene. A few investigators have demonstrated the possibility of aerosol transport of ethylbenzene from its source of emission to points substantially further downwind (>100 miles) (Sexton and Westberg, 1980a,b).

The most significant loss process for aquatic ethylbenzene is volatilization. The half-life for volatilization is estimated to range between <1 day and a few days (Lyman et al., 1982; Mabey et al., 1981). In summer months, however, when the temperature of water is higher than in other seasons, microbial degradation may be significant and may be competitive with the volatilization process (Gschwend et al., 1982). Significant removal of ethylbenzene through sorption onto particulate matters is not likely (Gschwend et al., 1982; Wakeham et al., 1983). Ethylbenzene will not significantly bioconcentrate in aquatic organisms (Nunes and Benville, 1979; Ogata et al., 1984).

Limited experimental data are available on the fate and transport of ethylbenzene in soil. Volatilization of ethylbenzene from soil surface is likely to be a significant loss process (Bouwer et al., 1984). Biodegradation of ethylbenzene in aerobic soil is likely to be competitive with the volatilization process (Bouwer et al., 1984; Kappeler and Wuhrmann, 1978a,b; Baltermann and Werner, 1984). From the measured concentrations of ethylbenzene at different soil depths at a waste site, Zoeteman et al. (1981) estimated the overall half-life of ethylbenzene in the disposal site to be ~37 days.

3. EXPOSURE

3.1. AIR

The atmospheric levels of ethylbenzene measured during the period 1973-1980 from 25 different sites in the United States, covering 11 rural/remote sampling points, 669 urban/suburban sampling points and 181 sampling points in source areas, were reported by Brodzinsky and Singh (1982). The median concentrations of ethylbenzene in rural/remote areas, urban/suburban areas and source areas were 470 ppt (2040 ng/m^3), 1200 ppt (5220 ng/m^3) and 630 ppt (2740 ng/m^3), respectively. The concentrations of ethylbenzene in the United States monitored during the 1980s and not reported by Brodzinsky and Singh (1982) are given in Table 3-1. The concentrations of ethylbenzene in a few cities outside of the United States are also shown in the same table.

From the measurement of ethylbenzene levels in 36 homes from the metropolitan Chicago, IL area, Jarke et al. (1981) reported that the frequency of occurrence of ethylbenzene was 57% in indoor air, compared with 32% in outdoor air. The mean concentration of ethylbenzene in indoor air in Manhattan, NY was 840 ppt (Potenta and Saunders, 1983). The mean concentration of ethylbenzene in the indoor air in Oak Ridge/Knoxville, TN, area was measured at 2090 ppt (Hawthorne et al., 1983). From the measurement of ethylbenzene levels in indoor and outdoor air of several locations, Wallace et al. (1983) reported that the median concentration value in indoor air is twice as high as the outdoor concentration. The higher value in indoor air is probably due to several factors, including the use of gas and oil for cooking and heating purposes, cigarette smoking and use of solvents containing ethylbenzene. Inadequate ventilation, particularly during the winter months, is responsible for any nonequilibrium of indoor and outdoor concentrations.

TABLE 3-1

Concentrations of Ethylbenzene in a Few U.S. and Foreign Locations in the 1980s

Location	Year Sampled	Mean Concentration (ppt)	Reference
	UNITED STATES		
Houston, TX (urban)	1980	1,380	Singh et al., 1981
St. Louis, MO (urban)	1980	640	Singh et al., 1981
Newark, NJ (urban)	1981	420[a]	Harkov et al., 1983, 1984
Elizabeth, NJ (urban)	1981	290[a]	Harkov et al., 1983, 1984
Camden, NJ (urban)	1981	200[a]	Harkov et al., 1983, 1984
Linville Gorge, NC (remote)	1981-1982	440	Seila et al., 1984
Rich Mountain, NC (remote)	1981-1982	330	Seila et al., 1984
Philadelphia, PA (urban)	1982	1,080	Haemisegger et al., 1985
Los Angeles, CA (urban)	1981	7,500[b]	Grosjean and Fung, 1984
	FOREIGN		
London, England (urban)	1982 1983	880 780	Clark et al., 1984a,b
Silwood Park, England (rural)	1982 1983	120 140	Clark et al., 1984a,b
Sidney, Australia	1979-1980	1,300	Nelson and Quigley, 1982

TABLE 3-1 (cont.)

Location	Year Sampled	Mean Concentration (ppt)	Reference
	FOREIGN (cont.)		
Middlesex, England (urban)	NA	16,100	Colenutt and Thorburn, 1980
The Hague, Netherlands (urban)	1974	5,000	Bos et al., 1977
Delft, Netherlands	1975	800	Bos et al., 1977

[a]These are mean values for summer and winter concentrations.

[b]Mean value of the concentration range given

NA = Not available

Since vehicular emission is one of the primary sources of ethylbenzene, the concentrations of ethylbenzene near motorways and tunnels are higher than ambient levels (Clark et al., 1984a,b; Perry and Twibell, 1973; Hampton et al., 1983; Kebbekus et al., 1983). For example, Kebbekus et al. (1983) reported the concentration of ethylbenzene to be 16,000 ppt in the Lincoln and Holland Tunnels, connecting New York to New Jersey compared with urban and rural ambient levels of 700 and 300 ppt, respectively. Emission of ethylbenzene from vehicles equipped with catalytic converters, however, were ~3 times lower than vehicles not so equipped (Hampton et al., 1983).

The levels of ethylbenzene in occupational situations have been reported by several investigators (Table 3-2), who have shown that ethylbenzene exposure from these sources can far exceed ambient air levels. Ethylbenzene has also been detected in the expired air of smokers (U.S. EPA, 1980) and in the blood of firefighters (Anderson et al., 1979). The mean value for post-1980 U.S. urban and rural atmospheric levels of ethylbenzene is estimated to be 1640 and 390 ppt, respectively (see Table 3-1). It is difficult to assess the change in atmospheric levels of ethylbenzene in the United States during the post-1980 period, compared with its pre-1980 values, with the limited available data for the post-1980 period; however, the level appears to have remained fairly constant. While introduction of the catalytic converter may have reduced ethylbenzene concentrations from vehicular emissions, the increase of aromatic content in unleaded fuel may have compensated for the expected reduction in ethylbenzene concentration. Since the pre-1980 data are based on much larger sampling sites, these values have been used for the estimation of inhalation exposure to ethylbenzene. Assuming that an individual inhales 20 m^3 of air/day and that the concentrations of ethylbenzene in rural/remote areas, urban/suburban areas and source

TABLE 3-2

Concentrations of Ethylbenzene in Occupational Situations

Operation	Concentration (ppb)	Reference
Rubber manufacturing plant	0-34.5	Cocheo et al., 1983
Paint and glues application	up to 800	Whitehead et al., 1984
Curing of synthetic rubber	22.7-28.8	Fraser and Rappaport, 1976
Rubber curing	53.8-98.0	Rappaport and Fraser, 1977
Histology laboratory using ethylbenzene	34,000-41,000	Angerer and Lehnert, 1979
Oil shale wastewater facility	1.6	Hawthorne and Sievers, 1984
Air over dumpsites	19-30	Levine et al., 1985
Fishmeal factory	5.1	Hoshika et al., 1981
Animal rendering factory	up to 800	Barnes and Macleod, 1982

areas are 2.04, 5.33 and 2.74 μg/m³, respectively, the corresponding daily inhalation exposures to the U.S. population are 41, 104 and 55 μg, respectively.

3.2. WATER

Ethylbenzene has been detected in industrial effluents, surface waters, groundwaters and in finished drinking water. Rawlings and Samfield (1979) reported the presence of ethylbenzene with a frequency of 63% in raw wastewater and with a frequency of 28% in secondary effluent water from textile plants. The concentration range of ethylbenzene in these samples was 0.7-3000 μg/ℓ. The concentration ranges of ethylbenzene in biological treatment effluent and final effluent from petroleum refining wastewater were <0.5 to >100 and <0.5 to <40 μg/ℓ, respectively (Snider and Manning, 1982). The concentration of ethylbenzene in water from a pump-out well at a SCM Corporation plant in Charlotte, NC, was reported to be 24 μg/ℓ (Kover, 1980a). Martin Marietta Chemicals reported the detection of ethylbenzene in well water adjacent to its plant sites. Unlined treatment lagoons were believed to be the source of this infiltration (Kover, 1980b). The concentration of ethylbenzene in final effluents from a Los Angeles County municipal wastewater treatment plant was reported to range between 34 and 220 μg/ℓ (Young et al., 1983).

Other investigators reported the detection of ethylbenzene in groundwater and leachates from contaminated sites. Kozloski and Sawhney (1982) reported the detection of ethylbenzene in leachates from a landfill site at Southington, CT. The concentration of ethylbenzene in groundwater at a depth of 13 m from a western Miami, FL (Miami Drum) waste disposal site was 5.2 μg/ℓ (Myers, 1983). Ethylbenzene concentrations ranging from 12-820 and 1.2-590 μg/ℓ were detected in leachates and groundwater from several municipal solid waste landfills in Minnesota (Sabel and Clark, 1984).

Stuermer et al. (1982) reported ethylbenzene in the concentration range of 92-400 µg/ℓ in three groundwater samples collected near two underground coal gasification sites in northeastern Wyoming. In The Netherlands, a concentration of up to 300 µg/ℓ ethylbenzene was detected in leachate from a waste dump site (Zoeteman et al., 1981). Well water from a landfill site in Waterloo, Ontario, contained as much as 98 µℓ/ℓ of ethylbenzene (Reinhard et al., 1984). Spillage of fuel near a public supply borehole in England contaminated the groundwater with ethylbenzene at a concentration of 1110 µg/ℓ in a site 160 m from the borehole (Tester and Harker, 1981). In a nationwide urban runoff program conducted by Cole et al. (1984), ethylbenzene was detected with a frequency of 4% and a concentration range of 1-2 µg/ℓ. Staples et al. (1985) examined the U.S. EPA's STORET data base and reported that ethylbenzene was detected in 7.4% of all effluent samples (total of 1368) at a median concentration of <3.0 µg/ℓ. In surface and groundwater samples, the frequency of occurrence of ethylbenzene was 10% and the median concentration for a total of 1101 samples was <5 µg/ℓ (Staples et al., 1985).

Ethylbenzene has also been reported in surface waters by other authors. The concentration of ethylbenzene in water from the Gulf of Mexico was reported to range between 0.4 and 4.5 ng/ℓ (Sauer et al., 1978). Other authors (Bertsch et al., 1975; Fielding et al., 1981; Malle, 1984) reported detection of ethylbenzene in surface waters. Goodley and Gordon (1976) reported the detection of 4.0 µg/ℓ of ethylbenzene in the lower Tennessee River.

The presence of ethylbenzene in drinking waters from Zurich, Switzerland (Grob, 1973), Evansville, IN (Kleopfer and Fairless, 1972), Canada (Otson et al., 1982), Philadelphia (Suffet et al., 1976) and Cincinnati (Kopfler et

al., 1975), and in charcoal-filtered drinking water (Dowthy et al., 1975) have been reported. A drinking water well near a landfill site in Jackson Township, NJ, contained 2000 µg/ℓ of ethylbenzene before the well was closed (Burmaster, 1982). The concentration of ethylbenzene in the Carrollton and Jefferson Parish, New Orleans, water plant was reported to range between 1.6 and 2.3 µg/ℓ (Keith et al., 1976). Ethylbenzene was detected at a concentration of 0.036 µg/ℓ in drinking water from Cincinnati, OH (Coleman et al., 1984). A drinking water well in Waverly, IA, was contaminated with 1 µg/ℓ of ethylbenzene; however, the compound was not detected in the finished water (Kelley, 1985). Ethylbenzene was detected with a frequency of 44% in 39 public water wells tested in New York State in 1979. The maximum ethylbenzene concentration was reported to be 40 µg/ℓ (CEQ, 1980). Water from only 0.8% of a total of 601 randomly and nonrandomly selected drinking water sites in the United States serving <10,000 people was reported to contain ethylbenzene at median and maximum concentrations of 0.9 and 1.0 µg/ℓ, respectively (Westrick et al., 1984). Similarly, water from ~0.3% of a total of 186 randomly and 158 non-randomly selected drinking water sites in the United States serving <10,000 people contained ethylbenzene at mean and maximum concentrations of 0.4 and 0.7 µg/ℓ, respectively (Westrick et al., 1984).

It is concluded from the preceding discussion that the vast majority of the people in the United States will not be exposed to significant amounts of ethylbenzene from the ingestion of drinking water. The concentration of ethylbenzene in drinking water in the United States will vary from 0-40 µg/ℓ. On the basis of an average consumption of 2 ℓ of drinking water/day, the daily exposure to ethylbenzene from drinking water will vary from 0-80 µg.

3.3. FOOD

Ethylbenzene was detected at a concentration of 0.8 ng/g (wet weight) in oysters from Lake Pontchartrain, New Orleans, LA (Ferrario et al., 1985). Tissues from aquatic animals collected near the discharge zone of the Los Angeles County wastewater treatment plant were reported to contain ethylbenzene, including the Pacific sanddab liver (<0.3 μg wet kg^{-1}), scorpion fish liver (<0.3 μg/ℓ wet kg^{-1}), dorr sole liver (0.3 μg wet kg^{-1}), croaker liver (4.0 μg wet kg^{-1}) and shrimp muscle (<0.3 μg wet kg^{-1}) (Gossett et al., 1983). Ethylbenzene has also been detected in cooked pork, salt pork and bacon; however, no quantitative value for ethylbenzene levels is available (Mottram et al., 1984). Roasted filbert nuts also were reported to contain unspecified amounts of ethylbenzene (U.S. EPA, 1980). Another possible source of ethylbenzene is food products packaged with styrene; although styrene has been detected in certain food products, the presence of ethylbenzene in these products has not been reported (U.S. EPA, 1980). Ethylbenzene has also been detected in mothers' milk in the New Jersey area with a frequency of 67% (Pellizzari et al., 1982). The origin of ethylbenzene in human milk may be from inhalation or ingestion sources or both.

Although ethylbenzene has been detected in a few food products, exposure to ethylbenzene from ingestion of food by the U.S. population cannot be estimated unless levels in composite food samples are known.

3.4. SUMMARY

The median concentrations of ethylbenzene in rural/remote areas, urban/suburban areas and source areas in the United States during 1973-1980 were 470 ppt (2040 ng/m^3), 1200 ppt (5220 ng/m^3) and 630 ppt (2740 ng/m^3), respectively (Brodzinsky and Singh, 1982). The concentrations of ethylbenzene in the United States during the 1980s remained fairly constant compared

with the previous years' values. Localized concentrations of ethylbenzene in enclosed high traffic density areas, such as tunnels, could be >20 times higher than ambient levels (Kebbekus et al., 1983). The level of ethylbenzene in occupational atmosphere may be as high as 41,000 ppb, as found in the histology laboratory (Angerer and Lehnert, 1979). Under certain conditions, the level of ethylbenzene in indoor air may be 2-3 times higher than outdoor air (Wallace et al., 1983; Hawthorne et al., 1983). Assuming that an individual inhales 20 m^3 of air/day and that the concentrations of ethylbenzene in rural/remote areas, urban/suburban areas and source areas are 2.04, 5.22 and 2.74 $\mu g/m^3$, respectively (Brodzinsky and Singh, 1982), the daily inhalation exposures to the U.S. population residing in these corresponding areas are 41, 104 and 55 µg, respectively.

Ethylbenzene has been detected in natural surface water, industrial effluents, treated municipal effluents, leachates from contaminated sites and in well waters. Staples et al. (1985) reported the detection of ethylbenzene in 7.4% of a total of 1368 effluent samples at a median concentration of <3.0 µg/ℓ. The surface and groundwater samples showed ethylbenzene with a frequency of 10% and a median concentration of <5 µg/ℓ. The concentration of ethylbenzene in U.S. drinking water can vary from 0-40 µg/ℓ. On the basis of an average consumption of 2 ℓ of drinking water/day, the daily exposure to ethylbenzene from drinking water will vary from 0-80 µg; however, the vast majority of the U.S. population will be exposed to the lowest dosage of ethylbenzene from ingestion of drinking water.

Although ethylbenzene has been detected in a few food products (Mottram et al., 1984; U.S. EPA, 1980), exposure to ethylbenzene from ingestion of food by the U.S. population cannot be estimated unless its levels in composite food samples are known.

4. PHARMACOKINETICS

4.1. ABSORPTION

After four female Wistar rats were treated with a single oral dose of 30 mg/kg of ring-labeled ^{14}C-ethylbenzene, ~80% of the total administered radioactivity was recovered from the urine collected for 24 hours (Climie et al., 1983). A total of ~82% of the radioactivity had been detected in the urine 48 hours after dosing, indicating that gastrointestinal absorption was extensive. Absorption of ethylbenzene from the GI tracts of rabbits was also extensive.

Groups of three male Harlan-Wistar rats were exposed to ring-labeled ^{14}C-ethylbenzene at a concentration of ~1 mg/ℓ (1000 mg/m^3) in an inhalation chamber for 6 hours (Chin et al., 1980). The mean quantity of ethylbenzene needed to maintain constant chamber pressure throughout the experiment and, therefore, the amount absorbed, was 16 mg/rat. If, as the investigators assumed, the rats inhaled 0.1 ℓ/minute, the total inhaled amount of ethylbenzene was 36 mg. Therefore, ~44% of the inhaled ethylbenzene was absorbed by the rats. The absorption rate of 44% includes whatever amount of ethylbenzene that was absorbed through the skin.

After 18 volunteers were exposed to ethylbenzene for 8 hours, at concentrations ranging from 23-85 ppm (100-370 mg/m^3), 64% of the inhaled ethylbenzene was "retained in the respiratory tract," presumably absorbed (Bardodej and Bardodejova, 1970).

4.2. DISTRIBUTION

In three male Wistar rats exposed to 1 mg/ℓ (1000 mg/m^3) of ^{14}C-ethylbenzene for 6 hours (see Section 4.1.), the amount of radioactivity found in the thyroid, adrenal, blood cells, lung, kidney, spleen, GI tract, liver, brain, fat or bone marrow at 48 hours after exposure was

<0.02% of the absorbed dose (Chin et al., 1980). The total in these tissues and the carcass was 0.199% of the absorbed dose. The liver had the highest percentage of the absorbed dose (0.014%), followed by the GI tract (0.008%), adipose tissue (0.007%) and lung (0.006%). No radioactivity was detected in the plasma.

Groups of five rats were killed after 2, 5, 9 and 16 weeks of exposure to 0, 50, 300 or 600 ppm (218, 1305 or 2610 mg/m^3) of ethylbenzene for 6 hours/day, 5 days/week, to analyze the ethylbenzene content of the perirenal fat (Engstrom et al., 1985). Although the concentration of ethylbenzene increased in the perirenal fat with increasing exposure levels, it did not increase with increasing exposure duration (Table 4-1); furthermore, it did not increase linearly with increasing dose levels.

Engstrom and Bjurstrom (1978) found that the concentration of ethylbenzene plus xylenes in the subcutaneous adipose tissue (6-7 mg/kg adipose tissue) of 12 male volunteers remained essentially constant between 2 and 22 hours after exposure to 100 or 200 ppm (435 or 870 mg/m^3) industrial xylene (40.4% ethylbenzene) for ~2 hours of rest and exercise. These results indicated that the solvent mixture was eliminated slowly from the tissue. The mean retention of ethylbenzene plus xylene at 22 hours represented ~5% of the total uptake.

4.3. METABOLISM

Analysis of the urine of four female Wistar rats given a single oral dose of 30 mg/kg of ring-labeled ^{14}C-ethylbenzene indicated that mandelic acid accounted for 23% of the administered dose and that hippuric acid accounted for 34% of the administered dose (Climie et al., 1983). Based on the urinary metabolites identified, the authors proposed the metabolic pathway for ethylbenzene indicated in Figure 4-1. 1-Phenylethylglucuronide was one of the minor metabolites identified.

TABLE 4-1

Concentration of Ethylbenzene in the Perirenal Fat of Rats After 2, 5, 9 and 16 Weeks of Exposure[a,b]

Exposure Level (ppm)	Ethylbenzene (mmol/g perirenal fat)			
	2 weeks	5 weeks	9 weeks	16 weeks
50	0.24±0.14	0.08±0.02	0.10±0.02	0.08±0.02
300	1.99±0.65	1.63±0.65	1.78±0.15	1.58±0.21
600	2.01±0.40	3.01±0.41	2.73±0.89	2.47±0.58

[a]Source: Engstrom et al., 1985

[b]Each figure denotes the means±SD of five rats; exposure was for 6 hours/day and 5 days/week.

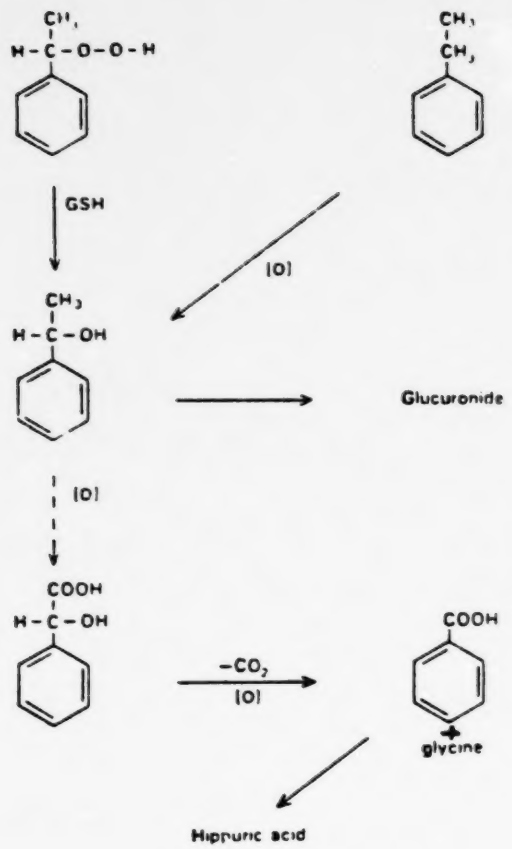


FIGURE 4-1

Metabolism of Ethylbenzene After Oral Administration to Female Rats

Source: Climie et al., 1983

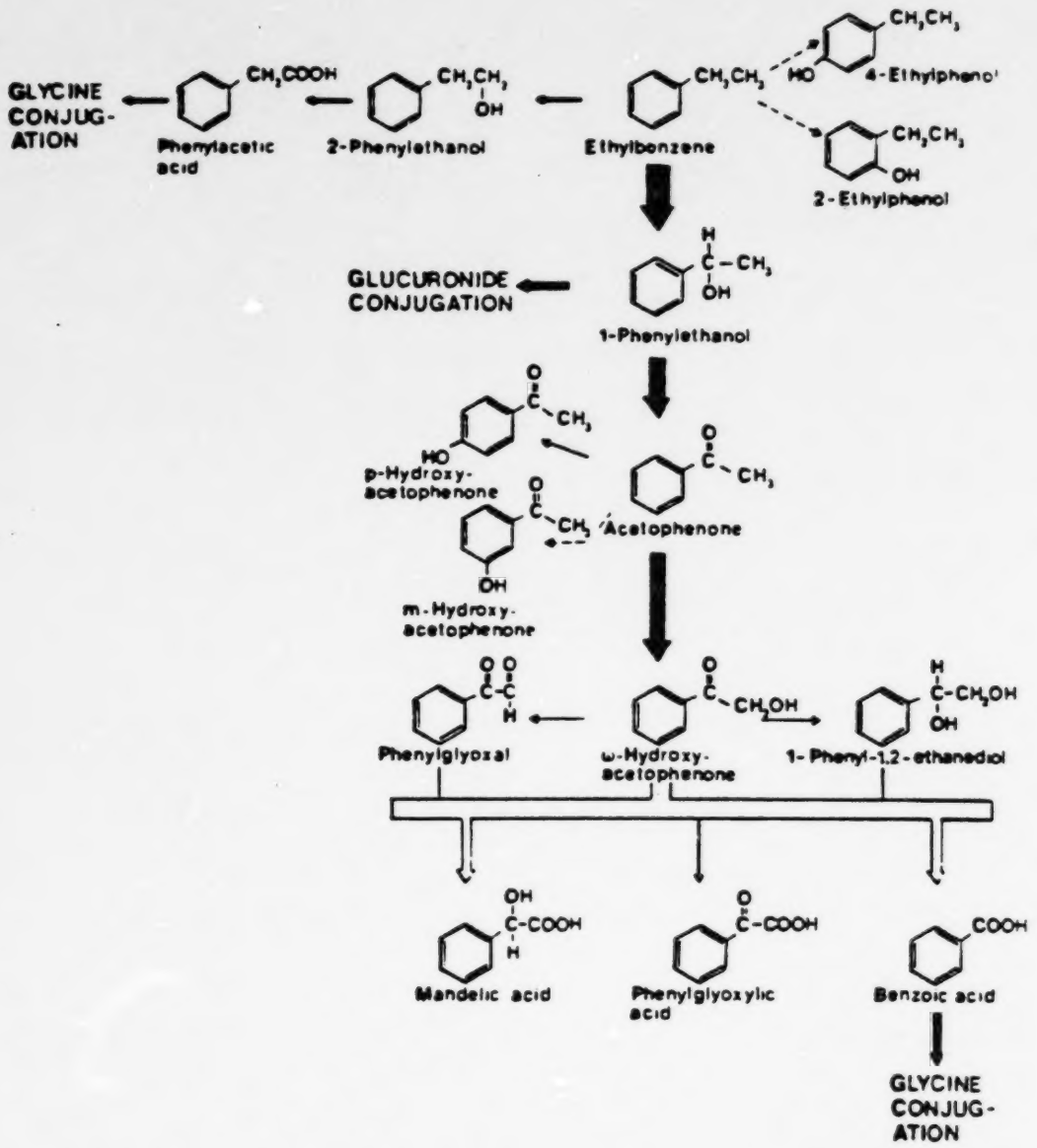
Hippuric acid and methylphenylcarbinyl glucosiduronic acid each accounted for ~30-35% of the dose of 3 mmol of ethylbenzene/kg administered by gavage to a group of three Chinchilla rabbits weighing 2.5-3.5 kg (El Masry et al., 1956). Phenaceturic acid and mandelic acid were also identified as urinary metabolites in the rabbits and accounted for 10-20% and 2% of the dose, respectively. Oxidation at either the α- or ω- carbon accounted for 70-90% of the metabolites identified in the urine of the rabbits after oral administration of ethylbenzene.

Engstrom (1984) proposed a metabolic pathway for ethylbenzene (Figure 4-2) based on the metabolites found in the urine (Table 4-2) of male Wistar rats exposed to $\leq$600 ppm ($\leq$2610 mg/m^3) ethylbenzene for a single 6-hour inhalation exposure (Engstrom, 1984) or for 6 hours/day, 5 days/week for up to 16 weeks (Engstrom et al., 1985).

Mandelic acid and phenylglyoxylic acid were the main urinary metabolites, respectively accounting for 64 and 25% of the retained dose, in volunteers exposed to 23-85 ppm (100-370 mg/m^3) of ethylbenzene for 8 hours (Bardodej and Bardodejova, 1970). Methylphenyl carbinol accounted for ~5% of the retained dose. Hagemann and Angerer (1979) found amygdalic acid, phenylglyoxylic acid and 2-ethylphenol in the urine of laboratory technicians occupationally-exposed to 34-42 ppm (148-183 mg/m^3) ethylbenzene. The English abstract of a Japanese study (Yamasaki, 1984) reported that after two volunteers had been exposed to 65 ppm (283 mg/m^3) of ethylbenzene for 3 hours, the three main urinary metabolites were mandelic acid, hippuric acid and phenylglyoxylic acid.

4.4. EXCRETION

In the Climie et al. (1983) study (Sections 4.1. and 4.3.), urine and feces of female rats were collected for 8 days after oral dosing with 30



The thickness of the arrows directly represents the extent of the respective route. Broken arrows mean that only trace amounts were found. Unclear pathways are depicted by open arrows.

FIGURE 4-2

Metabolic Pathway of Ethylbenzene in Rats

Source: Engstrom, 1984

TABLE 4-2

Percentage Composition of the Urinary Metabolite Pattern of Ethylbenzene at Two Exposure Levels*

Metabolite	Exposure Level	
	300 ppm	600 ppm
1-Phenylethanol	24.6	23.0
Mandelic acid	23.2	27.7
Phenylglyoxylic acid	9.9	10.9
Phenylacetic acid	7.3	6.6
Benzoic acid	27.0	23.9
ω-Hydroxyacetophenone	4.7	4.6
1-Phenyl-1,2-ethanediol	1.1	0.9
Acetophenone	0.2	0.1
p-Hydroxyacetophenone	0.5	0.7
Phenylglyoxal	1.4	1.2

*Source: Engstrom, 1984

mg/kg ^{14}C-ethylbenzene. No radioactivity was identified in either urine or feces after day 2. Of the administered radioactivity, 82% (80.3±7.6% on day 1 and 2.1±0.54% on day 2) was recovered from the urine and 1.5% of the administered radioactivity (1.1±0.46% on day 1 and 0.4±0.14% on day 2) was recovered from the feces.

As discussed in Sections 4.1. and 4.2., male rats were exposed to ^{14}C-ethylbenzene at a concentration of 1 mg/ℓ (1000 mg/m^3) for 6 hours, and the urine, feces and expired air were collected for the 48 or 72 hours following exposure (Chin et al., 1980). Excretion was essentially complete at 24 hours after exposure. A total of 91.7% of the administered radioactivity was recovered from the rats, with the greatest percentage being recovered from the urine (82.6%). Approximately 8% of the administered radioactivity was recovered from the expired air. The results are summarized in Table 4-3.

Groups of six male Wistar rats were exposed to 0, 300 or 600 ppm (0, 1305 or 2610 mg/m^3) of ethylbenzene for 6 hours during the nocturnal part of their day and urine was collected 6, 24, 30 and 48 hours after the start of treatment (Engstrom, 1984). In rats exposed to 300 ppm ethylbenzene, 83% of the absorbed dose was accounted for by urinary metabolites within 48 hours of dosing, with 6% of the absorbed dose accounted for during the 6-hour exposure period. The urinary metabolites, and their respective levels at the two dose levels tested, are listed in Table 4-2. The major urinary metabolites were identified as benzoic acid, mandelic acid and 1-phenylethanol, followed by phenylglyoxylic acid, phenylacetic acid and ω-hydroxyacetophenone. The other metabolites comprised ≥1% each of the metabolites identified.

TABLE 4-3

Recovery of Radioactivity During 24 Hours After Exposure of Rats to 1000 mg/m³ of ^{14}C-Ethylbenzene for 6 Hours[a,b]

Location	Absorbed Dose (%)
Urine	82.6 ±6.6
Feces	0.7 ±0.3
CO_2	0.03±0.01
Expired gases	8.2 ±3.8
Tissues	0.20±0.05
Total	91.7 ±2.6

[a]Source: Chin et al., 1980

[b]Mean and SD from two studies, three rats/study

Groups of ~20 male Wistar rats were exposed to 0, 50, 300 or 600 ppm (0, 218, 1305 or 2610 mg/m^3) of ethylbenzene for 6 hours/day, 5 days/week for up to 16 weeks (Engstrom et al., 1985). Urine samples were collected for 24 hours from two randomly selected rats from each group after 2, 5 and 9 weeks of treatment. As seen from Table 4-4, the total amount of ethylbenzene metabolites in the urine increased, though not linearly, with increasing exposure levels, but did not increase with increasing exposure duration. The percentages of the six main urinary metabolites changed with increasing exposure levels. The relative amounts of hippuric and benzoic acid decreased with increasing dose; the relative amount of phenylethanol increased with increasing dose; the relative amounts of mandelic, phenylglyoxylic and hippuric acids was decreased. Engstrom et al. (1985) emphasized that there was substantial individual variation in the excretion pattern of the separate metabolites.

As discussed in Section 4.3., 94% of the absorbed dose of ethylbenzene was recovered from the urine of volunteers exposed to 23-85 ppm (100-370 mg/m^3) of ethylbenzene for 8 hours (Bardodej and Bardodejova, 1970).

4.5. SUMMARY

At least 82% of the administered dose of ethylbenzene was absorbed from the GI tract after oral exposure in rats (Climie et al., 1983), whereas 44% and 64% of the administered dose of ethylbenzene was absorbed after inhalation exposure of rats (Chin et al., 1980) and humans (Bardodej and Bardodejova, 1970), respectively. Less than 0.2% of the administered radioactivity was recovered from the organs and carcasses of rats within 48-72 hours after inhalation exposure (Chin et al., 1980), indicating that substantial amounts of ethylbenzene are not retained. Furthermore, ethylbenzene did not accumulate with time in the perirenal fat of rats exposed to 50-600 ppm (218-2610 mg/m^3) of ethylbenzene for 6 hours/day, 5 days/week, for up to 16 weeks

TABLE 4-4

Urinary Excretion of Ethylbenzene[a,b]

	No. of Rats	Total Amount of Metabolites (μmol/24 hours)	Percentage of Total Amount of Metabolites Excreted					
			Mandelic Acid	Phenylglyoxylic Acid	Hippuric + Benzoic Acid	Phenaceturic + Phenylacetic Acid	1-Phenylethanol	ω-Hydryoxy-acetophenone
Dose (ppm								
50	14	72±41	18.9±7.8	14.8±10.1	47.8±18.9	4.2±3.6	12.4±7.4	4.6±3.2
300	14	256±56	19.6±4.2 (p<0.01)[c]	8.3±1.6 (p<0.05)[c,d]	39.6±7.5	3.5±2.5 (p<0.05)[c]	23.2±5.0 (p<0.001)[d]	6.4±2.9 (p<0.05)[c]
600	14	360±86	15.3±3.9	7.2±1.6 (p<0.01)[d]	37.6±7.4 (p<0.05)[d]	5.8±4.1	25.5±7.9 (p<0.001)[d]	8.4±1.9 (p<0.001)[d]
Exposure duration (weeks)								
2	12		21.0±8.4	10.8±5.3	38.3±15.3	3.5±4.2	22.8±8.6	5.8±3.5
5	12		17.5±3.9	10.5±9.3	45.5±13.0	5.3±2.8 (p<0.05)[e,f]	14.9±7.8	6.7±2.6
9	12		15.8±4.8 (p<0.05)[e]	9.2±7.2	40.8±13.0	5.6±3.5	21.8±9.8	7.8±2.2
Day of sampling								
Monday	24		16.4±3.4	9.3±5.0	43.0±10.9	5.5±3.8	19.4±7.9	6.7±3.1
Thursday	18		19.9±7.6 (p<0.05)[g]	11.2±8.6	39.6±15.5	3.4±0.5 (p<0.05)[g]	21.5±9.9	6.1±3.1

[a]Source: Engstrom et al., 1985

[b]The differences between the obtained values were statistically tested.

[c]Statistically different from values at 600 ppm

[d]Statistically different from values at 50 ppm

[e]Statistically different from values at 2 weeks

[f]Statistically different from values at 9 weeks

[g]Statistically different from values for Monday

0725p 02/26/86

(Engstrom et al., 1985). Approximately 82% of the administered radioactivity was recovered from the urine of rats treated with a single oral dose of ethylbenzene; 83% of the absorbed radioactivity was recovered from the urine of rats (Chin et al., 1980; Engstrom, 1984) and 94% from the urine of humans (Bardodej and Bardodejova, 1970) exposed to ethylbenzene by inhalation. In rats, only 1.5% of the orally administered dose (Climie et al., 1983) and 0.7% of the absorbed inhalation dose (Chin et al., 1980) was excreted in the feces. Excretion by the lungs of the absorbed inhalation dose was ~8% in rats (Chin et al., 1980). Mandelic (23%) and hippuric (34%) acid accounted for more than half of the radioactivity recovered from the urine of rats treated with a single oral dose of ethylbenzene (Climie et al., 1983). However, mandelic acid (23-28%), phenylethanol (23-25%) and benzoic acid (24-27%) accounted for ~75% of the radioactivity recovered from the urine of the rats after a single 6-hour inhalation exposure to ethylbenzene (Engstrom, 1984), indicating that the metabolic pathways are sensitive to the route of administration. With subchronic inhalation exposure to ethylbenzene, the ratio of the three major urinary metabolites in rats (mandelic acid, hippuric plus benzoic acid, and phenylethanol) was more sensitive to exposure dose than to exposure duration and did not appear to change over time; the possible exception was mandelic acid, which accounted for a statistically significant decreased ($p<0.05$) percentage of the total urinary metabolites after 9 weeks of treatment (Engstrom et al., 1985). After a single 8-hour inhalation exposure of humans to ethylbenzene, 64% of the retained dose was identified in the urine as mandelic acid and 25% as phenylglyoxylic acid (Bardodej and Bardodejova, 1970), indicating that there are species differences in the metabolism of ethylbenzene even when administered by the same route. There are species differences in the metabolism of

ethylbenzene after oral administration as well. Mandelic (23%) and hippuric (34%) acids were the major urinary metabolites after a single oral dose of ethylbenzene was administered to rats (Climie et al., 1983); however, methylphenylcarbinyl glucosiduronic (30-35%) and hippuric (30-35%) acids were the major urinary metabolites after a single oral dose of ethylbenzene was administered to rabbits (El Masry et al., 1956). Mandelic acid accounted for only 2% of the dose administered to rabbits.

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of ethylbenzene alone could not be located in the available literature as cited in the Appendix. Several epidemiological studies of occupational hazards in the production of styrene products, which indicate an increased risk of leukemias and lymphatic system cancer in workers, were reviewed by NCI (1985). Occupational exposure to styrene may occur in conjunction with ethylbenzene as well as benzene, butadiene, acrylonitrile and formaldehyde. Because of the confounding influence of the four latter chemicals, which are known carcinogens, conclusions regarding the carcinogenicity of styrene and ethylbenzene cannot be made (NCI, 1985). There are no data available for exposure to ethylbenzene per se and its potential carcinogenic effects.

The carcinogenicity testing of ethylbenzene has currently been deferred by the National Toxicology Program (NTP, 1985).

5.2. MUTAGENICITY

As indicated in Table 5-1, ethylbenzene had no mutagenic effect on Salmonella typhimurium strains TA98, TA100, TA1535, TA1537 or TA1538, with or without metabolic activation (Florin et al., 1980; Nestmann et al., 1980; Douglas et al., 1980; Dean et al., 1985). Similarly, ethylbenzene was not mutagenic in yeast (Nestman and Lee, 1983; Dean et al., 1985). Ethylbenzene was not clastogenic to rat liver cells in vitro (Dean et al., 1985). Ethylbenzene, however, increased the rate of SCE significantly in cultivated human lymphocytes at the highest dose tested, but not at lower doses, and therefore, Norppa and Vainio (1983) concluded that ethylbenzene was a very weak, noneffective mutagen.

Donner et al. (1980) reported that exposure of Drosophila melanogaster to ethylbenzene did not result in an increased incidence of recessive lethal

TABLE 5-1

Mutagenicity Testing of Ethylbenzene

Assay	Indicator Organism	Application	Concentration or Dose	Activating System	Response	Comment	Reference
Reverse mutation	Salmonella typhimurium TA98, TA100, TA1535, TA1537	plate incorporation	0.03, 0.3, 3.0 or 30.0 µmol/plate (0.003, 0.032, 0.319 or 3.185 mg/plate)	±S-9	=	30 µmol/plate was toxic to the bacteria.	Florin et al., 1980
Reverse mutation	S. typhimurium TA1535, TA100, TA1537, TA1538, TA98	plate incorporation	0.4 mg/plate	+S-9	-	The dose was the lowest that caused toxicity.	Nestmann et al., 1980; Douglas et al., 1980
Reverse mutation	S. typhimurium TA1535, TA1537, TA1538, TA98, TA100	plate incorporation	125-4000 µg/plate	±S-9	=	NC	Dean et al., 1985
Reverse mutation	Escherichia coli WP2, WP2 UVR A	plate incorporation	125-4000 µg/plate	±S-9	=	NC	Dean et al., 1985
Reverse mutation	Saccharomyces cerevisiae D7, XV18514C	exposure in liquid medium, then plate incorporation	NR	-S-9	-	The dose levels of exposure for the chemicals giving a negative response were not reported. The yeast was exposed to the chemical for 24 hours in liquid medium, then the cultures were plated and incubated for 4 days.	Nestmann and Lee, 1983
Mitotic conversion	S. cerevisiae JD1	plate incorporation	NR	±S-9	=	NC	Dean et al., 1985
Recessive Lethal	Drosophila melanogaster	NR	NR	NA	-	NC	Donner et al., 1980
Micronucleus test	mouse	oral dosing	NR	NA	-	NC	Fel'dt et al., 1985
SCE	human whole-blood lymphocytes	in liquid medium	0-10 mM solutions	none	see comment	Ethylbenzene induced a significant increase in the rate of SCE only at the highest dose tested. The authors concluded that ethylbenzene was not an effective mutagen.	Norppa and Vainio, 1983

NC = No comment; NR = not reported; NA = not applicable

0725p 02/26/86

mutations. Exposure to technical xylene that contains ~20% ethylbenzene, however, did significantly increase the incidence of recessive lethal mutations. No further details were given.

The abstract of a Russian study indicated that ethylbenzene was not mutagenic, as evaluated in the micronucleus test, after oral administration to mice (Fel'dt, 1985).

5.3. TERATOGENICITY

An inhalation teratology study conducted in New Zealand White rabbits was available as a brief summary by Hardin et al. (1981) and as a Battelle Laboratories technical report by Andrew et al. (1981). In this study, groups of 20-25 rabbits were exposed to 0, 100 or 1000 ppm (0, 435 or 4350 mg/m^3) ethylbenzene for 7 hours/day on days 1-24 of gestation. The rabbits were sacrificed on day 30 of gestation. Examination of the fetuses revealed no teratogenic effects. For the does, there were no effects on fertility, food consumption or body weight, no overt signs of toxicity and no differences in kidney, lung, spleen, ovary or uterus weights. The absolute and relative liver weights of the high-dose rabbits were significantly increased compared with controls; however, histological examination of the liver and other organs was unremarkable. Hardin et al. (1981) and Andrew et al. (1981) reported that the only effect on reproductive status was a statistically significant ($p<0.05$ by ANOVA) decrease in the number of live fetuses/litter. The number of live fetuses/litter (mean $\pm$ SD), however, were 8 ± 3, 7 ± 3 and 7 ± 3 for the control, low- and high-dose groups, respectively. The investigators stated the biological significance of the reduced number of live fetuses/litter was unclear because there were no differences in resorbed or dead fetuses/litter or preimplantation losses. The investigators concluded that ethylbenzene was not teratogenic or fetotoxic in this study.

Female Wistar rats were divided into seven groups of ~35 rats each (Hardin et al., 1981; Andrew et al., 1981). Two groups were exposed to 96 ppm [418 mg/m^3 (low)] and two groups were exposed to 959 ppm [4172 mg/m^3 (high)] for 7 hours/day, 5 days/week for 3 weeks before mating, while the remaining three groups were exposed to air only. After mating, the groups of rats were exposed to 0, 96 (low) or 985 (high) ppm of ethylbenzene for 7 hours/day on days 1-19 of gestation, in such a way as to provide seven different treatment groups, consisting of air-air, air-low, air-high, low-air, low-low, high-air and high-high exposed rats. The rats were sacrificed on day 21 of gestation. There was a significant increase in the liver, kidney and spleen weight in the pregnant rats treated at an air-high and high-high exposure level, but no associated histopathology was observed. Of the rats exposed to air during the 3 weeks before mating, 89% were pregnant but only 77% of the mated females exposed to ethylbenzene during the 3 weeks before mating were pregnant. Although the difference was statistically significant ($p<0.05$ by Chi square), no dose-related response was obvious (Table 5-2). No effect on number of corpora lutea, implantation sites, live fetuses/litter, dead fetuses/litter or litters with resorptions was reported. There was a significant ($p\leq0.05$, Fischer's Exact test) increase in the number of extra ribs in rats exposed to the high level of ethylbenzene during gestation (air-high and high-high groups) as well as in the air-low group. The biological significance of extra ribs is not clear and is considered a skeletal variant that may predict the potential for teratogenesis at higher exposure levels (Kimmel and Wilson, 1973). Andrew et al. (1981) reported a statistically significant decrease in crown-rump length of the air-high fetuses, but because this parameter was not decreased in the high-high fetuses, it was considered a spurious observation.

TABLE 5-2

Percent of Mated Female Rats that were Pregnant After Exposure to Ethylbenzene*

Treatment						
Air-Air	Air-Low	Air-High	Low-Air	Low-Low	High-Air	High-High
87	97	83	81	73	74	80

*Source: Andrew et al., 1981

Tatrai et al. (1982) reported that retarded skeletal development, increased incidence of extra ribs, depressed fetal weight gain and abnormal tail development were observed in rats exposed to 2400 mg/m³, but not 600 or 1200 mg/m³, of ethylbenzene for 24 hours/day on days 7-15 of pregnancy. Further details were not provided in the abstract of this Hungarian study.

5.4. OTHER REPRODUCTIVE EFFECTS

Germinal epithelial degeneration of the testes was observed in one rabbit and one monkey after 6 months inhalation exposure to ethylbenzene at 2.6 mg/ℓ (2600 mg/m³), 7 hours/day, 5 days/week (Wolf et al., 1956) (Section 5.5.). The histopathology was incompletely characterized.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Groups of 10 female rats were treated by gavage with 13.6, 136, 408 and 680 mg/kg/day of ethylbenzene in olive oil, 5 days/week for 6 months, for a total of 130 treatments in 182 days (Wolf et al., 1956). A group of 20 rats treated with olive oil served as controls. No effects on gross appearance, growth, hematology, BUN levels, bone marrow count, average final body or organ weight, or on the normal histology of lungs, heart, liver, kidneys, spleen, adrenals or pancreas were reported in the rats treated with 1.36 or 136 mg/kg/day. The rats treated with 408 or 680 mg/kg/day had increased liver and kidney weights accompanied by slight histopathological alterations, which were characterized by cloudy swelling of the liver parenchymal cells and of the renal tubular epithelium.

Groups of 10-25 male and female rats, 5-10 guinea pigs, 1-2 rabbits and 1-2 monkeys of either or both sexes were exposed by inhalation to ethylbenzene, 7 hours/day, 5 days/week for up to 6 months (Wolf et al., 1956). Matched controls were exposed to air only. The exposure levels and duration of exposure are summarized in Table 5-3.

The rats had increased liver and kidney weights at all exposure levels, but the increased liver and kidney weights were accompanied by histopathological alteration, characterized by cloudy swelling in both organs only at the 5.4 and 9.5 mg/ℓ (5400 and 9500 mg/m³) levels. The rats exposed to 5.4 mg/ℓ of ethylbenzene for 6 months had a questionable decrease in growth rate, whereas the rats exposed to the highest level of ethylbenzene had a slight decrease in growth rate.

No adverse effects were observed in the guinea pigs exposed to 1.7 mg/ℓ (1700 mg/m³). Increased liver weight was reported in the guinea pigs exposed to 2.6 mgℓ (2600 mg/m³) but not 5.4 mg/ℓ (5400 mg/m³) of ethylbenzene. There was a slight depression of growth rate in the guinea pigs exposed to 5.4 mg/ℓ of ethylbenzene. No adverse histological changes were reported in the guinea pigs at any exposure level tested.

No effect was observed in the rabbits exposed to 1.7 mg/ℓ (1700 mg/m³) of ethylbenzene. Testicular histopathology characterized by germinal epithelial degeneration was reported in one rabbit exposed to 2.6 mg/ℓ (2600 mg/m³) of ethylbenzene. Only female rabbits were exposed to 5.4 mg/ℓ (5400 mg/m³) and no adverse effects were reported.

Monkeys were exposed to two levels of ethylbenzene. At 1.7 mg/ℓ (1700 mg/m³), no adverse effects were reported. Slightly increased liver weight and testicular histopathology, characterized by germinal epithelial degeneration, was observed in the monkey exposed to 2.6 mg/ℓ (2600 mg/m³) of ethylbenzene.

All the changes reported in rats, guinea pigs, rabbits and monkeys treated with ethylbenzene were considered slight, except for the increased kidney weight in rats exposed to the 9.5 mg/ℓ, which was considered moderate. No statistical analysis of the data were performed and histopathology was incompletely reported.

TABLE 5-3

Summary of Ethylbenzene Exposure Levels, Duration of Exposure and Results in Rats, Guinea Pigs, Rabbits and Monkeys*

Species	Average Vapor Concentrations (mg/ℓ)	Sex	Number of 7-Hour Exposures	Duration of Experiments (days)
Rat	9.5	M	103	144
	5.4	M,F	138	214
	2.6	M,F	130	186
	1.7	M,F	130	186
Guinea pig	5.4	F	138	214
	2.6	M,F	130	186
	1.7	M,F	130	186
Rabbit	5.4	F	138	214
	2.6	M,F	130	186
	1.7	M,F	130	186
Rhesus monkey	2.6	M,F	130	186
	1.7	F	130	186

*Source: Wolf et al., 1956

Groups of 20 male Wistar rats were exposed to 0, 50, 300 or 600 ppm (0, 218, 1305 or 2610 mg/m³) of ethylbenzene (99% purity) for 6 hours/day, 5 days/week and killed after 2, 5, 9 or 16 weeks of exposure (five/dose/kill) (Elovaara et al., 1985). In addition to light and electron microscopic examination of the liver, SGPT levels, hepatic NADPH-cytochrome c reductase, aminopyrine N-demethylase, propionaldehyde dehydrogenase and D-glucurono-lactone dehydrogenase levels, renal glutathione levels, and hepatic and renal microsomal protein, cytochrome P-450, ethoxycoumarin O-deethylase and UDP-glucuronosyl-transferase levels in both kidney and liver were determined. After 16 weeks of exposure, no biologically significant effect was observed on the levels of SGPT, hepatic and renal microsomal protein or cytochrome P-450, hepatic NADPH-cytochrome c reductase or propionaldehyde. The levels of all the other enzymes had increased at the two highest dose levels. No morphological alterations in the liver were observed with the light microscope. Electron microscopy revealed changes normally seen after introduction of any xenobiotics, including proliferation of smooth endoplasmic reticulum and altered mitochondria, which were no longer detectable after 16 weeks of treatment at all exposure levels except the highest (600 ppm).

5.6. OTHER RELEVANT INFORMATION

The acute oral LD_{50} values of ethylbenzene were reported to be 3.5 g/kg for adult Wistar rats of both sexes (Wolf et al., 1956) and 5.46 mℓ/kg (4.7 g/kg) for male rats (Smyth et al., 1962).

Ethylbenzene constitutes ~20% of a standard xylene solution and makes identification of effects produced exclusively by xylene or ethylbenzene impossible to identify. Angerer and Lehnert (1979) reported that ethylbenzene competitively inhibits ring oxidation of xylene when present simultaneously. Both xylene and ethylbenzene increased P-450 concentrations,

NADPH-cytochrome c reductase, 7-ethoxyresorufin O-deethylase and hexane hydroxylase activity (Toftgaard and Nilsen, 1981, 1982). Exposure to mixtures of xylenes and ethylbenzene affects the CNS in humans (Gamberale et al., 1978; Vol'Fovskaya and Makulova, 1971), cats (Carpenter et al., 1975) and rats (Andersson et al., 1981; Tham et al., 1984).

5.7. SUMMARY

Pertinent data regarding the carcinogenicity of ethylbenzene alone could not be located in the available literature as cited in the Appendix. Studies of hazards to workers employed in the production of styrene products, which may involve exposure to ethylbenzene, are confounded by concurrent exposure to such known carcinogens as benzene, butadiene, acrylonitrile and formaldehyde (NCI, 1985). Ethylbenzene was not mutagenic in bacteria (Florin et al., 1980; Hestman et al., 1980; Douglas et al., 1980), yeast (Nestman and Lee, 1983) or Drosophila melanogaster (Donner et al., 1980), but was weakly mutagenic to human lymphocytes in vitro, as evaluated by the SCE assay (Norppa and Vainio, 1983). Ethylbenzene had no significant adverse effect on fetal development and was not teratogenic to rabbits at inhalation concentrations $\leq$1000 ppm ($\leq$4350 mg/m^3), 7 hours/day during gestation (Hardin et al., 1981; Andrew et al., 1981). Rat fetuses had increased incidences of extra ribs following exposure of dams to 96 or 959 ppm (418 or 4172 mg/m^3), 7 hours/day during gestation. Increased liver and kidney weights accompanied by hepatic and renal histopathology (cloudy swelling) were observed in rats treated orally with $\geq$408 mg/kg/day of ethylbenzene, 5 days/week for 6 months (Wolf et al., 1956). After inhalation exposure for 6 months, similar hepatic and renal effects were observed in rats exposed to $\geq$5400 mg/m^3, 7 hours/day, 5 days/week (Wolf et al., 1956).

Increased liver and kidney weight also occurred at ≥1700 mg/m³, 7 hours/day, 5 days/week. After 6 months of inhalation exposure, guinea pigs had slightly decreased growth rates at 5400 mg/m³ and slightly increased liver weight at 2600 mg/m³, 7 hours/day, 5 days/week (Wolf et al., 1956). Testicular changes characterized by germinal epithelial degeneration were observed in rabbits and monkeys at 2600 mg/m³, 7 hours/day, 5 days/week (Wolf et al., 1956). The monkeys also had slightly increased liver weights. Hepatic and renal enzyme levels were increased in rats exposed by inhalation to 300 or 600 ppm (1305 or 2610 mg/m³) ethylbenzene, 6 hours/day, 5 days/week for up to 16 weeks (Elovaara et al., 1985).

6. AQUATIC TOXICITY

6.1. ACUTE

The available information about acute toxicity of ethylbenzene to freshwater and saltwater fishes is presented in Table 6-1. The most sensitive species tested was the striped bass, Morone saxatilis, with a 96-hour LC_{50} of 3.7 mg/ℓ (Benville and Korn, 1977). The least sensitive species was the sheepshead minnow, Cyprinodon variegatus, with a 96-hour LC_{50} of 0.275 mg/ℓ (U.S. EPA, 1978).

There is a great deal of variability in reported toxic concentrations for invertebrates (Table 6-2). Reported LC_{50} values for Daphnia magna range from 2.12 (Bobra et al., 1983) to 190 mg/ℓ (Bringmann and Kuehn, 1977). The most sensitive species tested was the bay shrimp, Crago franciscorum, with a 96-hour LC_{50} of 0.42 mg/ℓ (Benville and Korn, 1977). The least sensitive species tested was the pacific oyster, Crassoatrea gigas, with a LC_{50} value of 1030 mg/ℓ (LeGore, 1974).

6.2. CHRONIC

There is little information concerning chronic toxicity of ethylbenzene to aquatic organisms. In an embryo-larval test with fathead minnows, Pimephales promelas, no adverse effects occurred at 0.44 mg/ℓ, the highest concentration used (U.S. EPA, 1978). Kenaga (1982) reported that the acute/chronic toxicity ratio for ethylbenzene was <110 for fathead minnows.

6.3. PLANTS

Hutchinson et al. (1980) determined that the concentrations that caused a 50% inhibition of photosynthesis in algae were 51 mg/ℓ for Chlamydomonas angulasa and 63 mg/ℓ for Chlorella vulgaris. U.S. EPA (1978) determined that EC_{50} values for decreases in cell numbers or chlorophylla were >438 mg/ℓ for Selenastrum capricornutum and Skeletonema costatum. Bringmann

TABLE 6-1

Acute Toxicity of Ethylbenzene to Fishes

Species	Concentration (mg/ℓ)	Effect	Reference
		FRESHWATER	
Bluegill	150	96-hour LC_{50}	Buccafusco et al., 1981
Lepomis macrochirus	32	96-hour LC_{50}	Pickering and Henderson, 1966
	155	96-hour LC_{50}	U.S. EPA, 1978
	36	NOEC	U.S. EPA, 1978
Fathead minnow	48.5	24-, 48-, 96-hour LC_{50}, soft water	Pickering and Henderson, 1966
Pimephales promelas	42.3	24-, 48-, 96-hour LC_{50}, hard water	Pickering and Henderson, 1966
Goldfish	94.4	24-, 48-, 96-hour LC_{50}	Pickering and Henderson, 1966
Carassius auratus			
Golden orfe	26	48-hour LC_0	Juhnke and Luedemann, 1978
Leuciscus idus	44	48-hour LC_{50}	Juhnke and Luedemann, 1978
	70	48-hour LC_{100}	Juhnke and Luedemann, 1978
Guppy	97.1	96-hour LC_{50}	Pickering and Henderson, 1966
Poecilia reticulata			
Coho salmon	0	6.7% mortality, 96 hours	Morrow et al., 1975
Oncorhynchus kisutch	10	13.3% mortality, 96 hours	Morrow et al., 1975
	50	100% mortality, 96 hours	Morrow et al., 1975

TABLE 6-1 (cont.)

Species	Concentration (mg/ℓ)	Effect	Reference
		SALTWATER	
Striped bass Morone sazatilis	3.7	96-hour LC_{50}	Benville and Korn, 1977
Sheepshead minnow Cyprinodon variegatus	305	24-hour LC_{50}	U.S. EPA, 1978
	361	48-hour LC_{50}	U.S. EPA, 1978
	321	72-hour LC_{50}	U.S. EPA, 1978
	275	96-hour LC_{50}	U.S. EPA, 1978
	87.6	NOEC	U.S. EPA, 1978

TABLE 6-2

Acute Toxicity of Ethylbenzene to Invertebrates

Species	Concentration (mg/ℓ)	Effect	Reference
		FRESHWATER	
Waterflea *Daphnia magna*	75	48-hour LC_{50}	U.S. EPA, 1978
	6.8	48-hour NOEC	U.S. EPA, 1978
	120	24-hour LC_0	Bringmann and Kuehn, 1977
	190	24-hour LC_{50}	Bringmann and Kuehn, 1977
	200	24-hour LC_{100}	Bringmann and Kuehn, 1977
	137	24-hour EC_0, immobilization	Bringmann and Kuehn, 1982
	184	24-hour EC_{50}, immobilization	Bringmann and Kuehn, 1982
	200	24-hour EC_{100}, immobilization	Bringmann and Kuehn, 1982
	2.12	48-hour LC_{50}	Bobra et al., 1983
Protozoan *Entosiphon sulcatum*	140	toxicity threshold, inhibition of cell multiplication	Bringmann and Kuehn, 1980
		SALTWATER	
Bay shrimp *Crago franciscorum*	0.42	96-hour LC_{50}	Benville and Korn, 1977
Grass shrimp *Palaemonetes pugio*	10.2	24-hour LC_{50}, larvae	Potera, 1975
	14.4-17.3	24-hour LC_{50}, adults	Potera, 1975
Mysid shrimp *Mysidopsis bahia*	88	96-hour LC_{50}	U.S. EPA, 1978
	<43.8	NOEC	U.S. EPA, 1978
Copepod *Nitocra spinipes*	16	24-hour LC_{50}	Potera, 1975
Pacific oyster *Crassostrea gigas*	1030	LC_{50}	LeGore, 1974

and Kuehn (1978) reported toxicity thresholds for inhibition of cell multiplication of >160 mg/ℓ in the green algae, Scenedesmus quadricauda, and 33 mg/ℓ in the blue-green algae, Microcystis aeruginosa. The toxicity threshold was 12 mg/ℓ for the bacterium, Pseudomonas putida (Bringmann and Kuehn, 1976).

6.4. RESIDUES

Pertinent data regarding ethylbenzene residues in aquatic biota could not be located in the available literature as cited in the Appendix. U.S. EPA (1983) calculated an estimated BCF of 150 for aquatic organisms, based on a K_{ow} of 1400.

6.5. SUMMARY

There is limited information concerning toxicity of ethylbenzene to aquatic organisms. Acutely toxic concentrations range from 0.42 mg/ℓ for the bay shrimp (Benville and Korn, 1977) to 1030 mg/ℓ for the Pacific oyster (LeGore, 1974). The most sensitive plant species tested was the blue-green algae, Microcystis aeruginosa, with a toxicity threshold of 33 mg/ℓ (Bringmann and Kuehn, 1978), while the bacterium, Pseudomonas putida, had a toxicity threshold of 12 mg/ℓ (Bringmann and Kuehn, 1976).

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

To protect against headache, fatigue and irritation of mucous membranes, ACGIH (1980) has recommended and ACGIH (1985) has adopted a TLV of 100 ppm (435 mg/m^3), with a STEL of 125 ppm (545 mg/m^3). The OSHA (1985) standard for occupational exposure to ethylbenzene is 100 ppm (435 mg/m^3).

An ADI of 0.097 mg/kg/day, or 6.8 mg/day for a 70 kg human, was reported by the U.S. EPA (1985a,b).

7.2. AQUATIC

U.S. EPA (1980) did not derive a criterion for the protection of aquatic life from the effects of ethylbenzene; however, it did state that acute toxicity to freshwater and saltwater organisms occurred at concentrations as low as 32 and 0.43 mg/ℓ, respectively, and would occur at lower concentrations in species more sensitive than those tested.

8. RISK ASSESSMENT

Pertinent data regarding the carcinogenicity of ethylbenzene alone could not be located in the available literature as cited in the Appendix. Studies of hazards to workers employed in the production of styrene products, which may involve exposure to ethylbenzene, are confounded by concurrent exposure to such known carcinogens as benzene, butadiene, acrylonitrile and formaldehyde (NCI, 1985). Although ethylbenzene was not mutagenic to bacteria (Florin et al., 1980; Nestman et al., 1980; Douglas et al., 1980; Dean et al., 1985), yeast (Nestman and Lee, 1983; Dean et al., 1985), or Drosophila melanogaster (Donner et al., 1980), it did increase the incidence of SCE in cultivated human lymphocytes (Norppa and Vainio, 1983). Ethylbenzene had no significant adverse effect on fetal development and was not teratogenic to rabbits at inhalation exposures $\leq$1000 ppm ($\leq$4350 mg/m^3), 7 hours/day during gestation (Hardin et al., 1981; Andrew et al., 1981). Rat fetuses had increased incidences of extra ribs following exposure of dams to 96 or 951 ppm (418 or 4172 mg/m^3), 7 hours/day during gestation. No effects were observed in rats treated orally with $\leq$136 mg/kg/day of ethylbenzene for 5 days/week, for 6 months (Wolf et al., 1956). At $\geq$408 mg/kg/day, 5 days/week, the rats had increased liver and kidney weights and cloudy swelling in these organs. Increased liver and kidney weights were observed in rats exposed by inhalation to $\geq$1700 mg/m^3 for 7 hours/day, 5 days/week for 6 months, and renal and/or hepatic histopathology (cloudy swelling) was present in rats exposed to $\geq$5400 mg/m^3, 7 hours/day, 5 days/week (Wolf et al., 1956). After 6 months of inhalation exposure, guinea pigs had a slightly decreased growth rate at 5400 mg/m^3 and slightly increased liver weights at 2600 mg/m^3, 7 hours/day, 5 days/week

(Wolf et al., 1956). Testicular changes characterized by germinal epithelial degeneration were observed in rabbits and monkeys exposed to 2600 mg/m^3, 7 hours/day, 5 days/week for 6 months. The monkeys also had slightly increased liver weights. Hepatic and renal enzyme levels were increased in rats exposed by inhalation to 300 or 600 ppm (1305 or 2610 mg/m^3), 6 hours/day, 5 days/week for up to 16 weeks (Elovaara et al., 1985).

As reported in U.S. EPA (1985a,b), the study of Wolf et al. (1956), in which rats were treated by gavage with 13.6, 136, 408, or 680 mg/kg/day of ethylbenzene, 5 days/week for 6 months, provides the most appropriate data from which to derive an ADI. The highest dose at which no adverse effects were observed is 136 mg/kg/day, since increased liver and kidney weights accompanied by hepatic and renal histopathology were reported at the next highest dose tested (408 mg/kg/day). The animal NOEL of 97 mg/kg/day is obtained by multiplying the administered dose of 136 mg/kg/day by 5/7 to convert it to continuous exposure. An ADI of 0.097 mg/kg/day (6.8 mg/day for a 70 kg human) was obtained from the animal NOEL by dividing by an uncertainty factor of 1000 to account for interspecies extrapolation, the range of sensitivity within the human population to xenobiotics, and the use of a subchronic study to derive an ADI.

9. REPORTABLE QUANTITIES

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

Chronic oral or inhalation studies of ethylbenzene were not available; however, a 6-month oral study in rats, a 6-month inhalation study in several species (Wolf et al., 1956), a 16-week inhalation study using rats (Elovaara et al., 1985) and two inhalation teratology studies using rats (Hardin et al., 1981; Andrew et al., 1981) described effects that can be considered in the derivation of an RQ for ethylbenzene. These studies were discussed in Chapter 5 and are summarized in Table 9-1.

As seen from Table 9-1, effects of ethylbenzene exposure are increased liver and kidney weights, depressed growth rate, cloudy swelling of cells in the liver and kidney, enzyme induction, degeneration of germinal epithelial cells in the testes and slight evidence of fetotoxicity (extra ribs). The lowest transformed dose at which liver and kidney weights were increased, but no effects of greater severity occurred, were 226 mg/kg/day for rats and 258 mg/kg/day for guinea pigs in the inhalation study of Wolf et al. (1956). Multiplying the doses by the cube root of the ratio of the animal body weight to the reference human body weight results in equivalent human doses of 38.6 and 59.1 mg/kg/day, respectively. Multiplying the lower value of 38.6 mg/kg/day by 70 kg, and dividing by an uncertainty factor of 10 to approximate chronic exposure, results in an MED of 270 mg/day. The MED corresponds to an RV_d of 1.9. The RV_e for increased organ weights without histolopathological lesions is 4. Multiplying the RV_d by the RV_e results in a CS of 7.6, which corresponds to an RQ of 1000 (Table 9-2).

Cloudy swelling of the liver and kidney occurred in rats at transformed doses $\geq$291 mg/kg/day in the oral study and at $\geq$717 mg/kg/day in the inhalation study by Wolf et al. (1956). Multiplying the lower dose by the cube

TABLE 9-1

Toxicity Summary for Ethylbenzene

Route	Species/ Strain	Sex	No. at Start	Average Weight Assumed (kg)	Vehicle/ Physical State	Purity	Exposure	Transformed Animal Dose (mg/kg/day)	Response	Reference
Oral (gavage)	rat/Wistar	F	10/group	0.35[a]	olive oil	>98%	408 or 680 mg/kg/day, 5 days/week for 6 months	291 or 486[b]	Slightly increased liver and kidney weight accompanied by cloudy swelling	Wolf et al., 1956
Inhalation	rat/Wistar	F	35/group	0.27[c]	vapor	pure grade	96 or 959 ppm (418 or 4712 mg/m^3) 7 hours/ day during gestation	85 or 846[d]	Increased incidence of extra ribs in fetuses	Andrew et al., 1981; Hardin et al., 1981
Inhalation	rat/Wistar	M,F	10-25 group	0.35[a]	vapor	>98%	1700, 2600, 5400 or 9500 mg/m^3, 7 hours/day, 5 days/week for ~6 months	226, 345, 717 of 1261[d]	Increased liver and kidney weight at $\geq$226 mg/kg/day; cloudy swelling in liver and kidney at $\geq$717 mg/kg/ day, moderately de- creased growth rate at 1261 mg/kg/day	Wolf et al., 1956
Inhalation	rat/Wistar	M	20/group	0.35[a]	vapor	99%	300 or 600 ppm (1305 or 2610 mg/m^3) 6 hours/ day, 5 days/week up to 16 weeks	148 or 297[d]	Induction of liver and kidney enzymes	Elovaara et al., 1985
Inhalation	guinea pig/albino	M,F	5-10/group	0.84[e]	vapor	>98%	2600 or 5400 mg/m^3, 7 hours/ day for ~6 months	258 or 536[f]	Increased liver weight at 258 mg/kg/day; de- creased growth rate at 536 mg/kg/day	Wolf et al., 1956
Inhalation	rabbit/ albino	M,F	1-2	3.8[e]	vapor	>98%	2600 mg/m^3, 7 hours/day, 5 days/week for ~6 months	285[f]	Degeneration of germi- nal epithelium in the testes of males	Wolf et al., 1956

TABLE 9-1 (cont.)

Route	Species/ Strain	Sex	No. at Start	Average Weight Assumed (kg)	Vehicle/ Physical State	Purity	Exposure	Transformed Animal Dose (mg/kg/day)	Response	Reference
Inhalation	monkey/ rhesus	M,F	2	8.0[e]	vapor	>98%	2600 mg/m³, 7 hours/day, 5 days/week for ~6 months	366[f]	Increased liver weight; degeneration of germinal epithelium in testes of males	Wolf et al., 1956

[a]Assumed reference body weight (U.S. EPA, 1980)

[b]The transformed dose was calculated by multiplying the experimental dose by 5 days/7 days.

[c]Mean body weight of nongravid rats in the study

[d]The doses were calculated by multiplying the exposure by 7 hours/24 hours, by 5 days/7 days, and by the rat inhalation rate, and by dividing by the rat body weight. The inhalation rate was calculated using the expression $0.105(\text{rat weight}/0.113)^{2/3}$ (U.S. EPA, 1980).

[e]Assumed reference body weight from Durkin (1985)

[f]Calculated by expanding to continuous exposure and multiplying the inhalation rate and dividing by the body weight. Inhalation rates were 0.40 m³/day for guinea pigs, 2.0 m³/day for rabbits and 5.4 m³/day for monkeys.

0725p

02/26/86

TABLE 9-2

Composite Scores for Ethylbenzene

Route	Species	Animal Dose (mg/kg/day)	Chronic Human MED (mg/day)	RV_d	Effect	RV_e	CS	RQ	Reference
Oral	rat	291	348*	1.7	Increased liver and kidney weights accompanied by cloudy swelling	5	8.5	1000	Wolf et al., 1956
Inhalation	rabbit	285	755*	1.2	Slight degeneration of the germinal epithelium in the testes	6	7.2	1000	Wolf et al., 1956
Inhalation	rat	226	270*	1.9	Increased liver and kidney weights	4	7.6	1000	Wolf et al., 1956
Inhalation	rat	148	177*	2.1	Enzyme induction	1	2.1	5000	Elovaara et al., 1985
Inhalation	rat	85	933	1.0	Fetotoxicity	8	8	1000	Andrew et al., 1981; Hardin et al., 1981

*The dose was dividied by an uncertainty factor of 10 to approximate chronic exposure.

root of the body weight ratio, and by 70 kg, and dividing by an uncertainty factor of 10, results in an MED of 348 mg/day. The RV_d is 1.7, the RV_e is 5 and the CS is 8.5, which corresponds to an RQ of 1000.

Decreased body weight, which warrants an $RV_e \leq 4$, occurred at higher doses; therefore, no MED calculation for this effect is necessary.

Liver and kidney enzymes were induced in rats at ≥148 mg/kg/day in the inhalation study of Elovaara et al. (1985). The MED is 177 mg/kg/day, which corresponds to an RV_d of 2.1. The RV_e is 1 and the CS is 2.1, which corresponds to an RQ of 5000.

Degeneration of the germinal epithelium of the testes occurred in rabbits at 285 mg/kg/day and in monkeys at 366 mg/kg/day. The use of the rabbit dose in the calculation results in the lower MED of 755 mg/day, which corresponds to an RV_d of 1.2. An uncertainty factor of 10 was used. Testicular degeneration warrants an RV_e of 6. Multiplying the RV_d by the RV_e results in a CS of 7.2, corresponding to an RQ of 1000.

Evidence of fetotoxicity was observed in rat fetuses at ≥85 mg/kg/day (Andrew et al., 1981; Hardin et al., 1981). Multiplying the dose by the cube root of the ratio of the rat weight (0.27 kg) to the human weight of 70 kg and by 70 kg results in an MED of 933 mg/day. No uncertainty factor is needed because the exposure was chronic to the life of the fetus. The RV_d is 1.0, the RV_e is 8 and the CS is 8, which corresponds to an RQ of 1000.

As can be seen from Table 9-2, the highest CS was obtained using the data for increased liver and kidney weight accompanied by cloudy swelling in the hepatocytes and in the renal tubular epithelium in rats administered ethylbenzene by gavage at a transformed dose of 219 mg/kg/day and, therefore, form the basis for the RQ of 1000 for ethylbenzene (Table 9-3).

TABLE 9-3

Ethylbenzene

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:	oral
Dose*:	348 mg/day
Effect:	Increased liver and kidney weight accompanied by histological alterations
Reference:	Wolf et al., 1956
RV_d:	1.7
RV_e:	5
Composite Score:	8.5
RQ:	1000

*Equivalent human dose

In a previous determination of the RQ for ethylbenzene, the U.S. EPA (1983) used the data of Wolf et al. (1956) for increased liver and kidney weights in rats at inhalation exposures $\geq$1700 mg/m^3, 7 hours/day, 5 days/week, as the basis for an RQ of 5000 for ethylbenzene. There are several reasons for the differences between the RQ determination by U.S. EPA (1983) and the present determination. First, because the original paper was not available, U.S. EPA (1983) disregarded the oral part of the study by Wolf et al. (1956), which provides the highest CS. Second, the methodology in U.S. EPA (1983) converted animal inhalation to human dose levels by using human breathing rates rather than the animal inhalation rates and the cube root of the body weight ratio, resulting in different CSs. And third, the human chronic MED was obtained by dividing the subchronic exposure data by a factor of 5 rather than the 10 that was used to obtain the RQ in this document. Given the lack of any chronic studies, it seems more appropriate to use the higher uncertainty factor.

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

Pertinent data regarding the carcinogenicity of ethylbenzene alone could not be located in the available literature as cited in the Appendix. Several epidemiological studies of occupational hazards in the production of styrene products, which indicated an increased risk of leukemias and lymphatic system cancers in workers, were reviewed by NCI (1985). Occupational exposure to styrene may occur in conjunction with ethylbenzene, as well as benzene, butadiene, acrylonitrile and formaldehyde. Because of the confounding influence of the four latter chemicals, which are known carcinogens, conclusions regarding the carcinogenicity of styrene or ethylbenzene could not be made (NCI, 1985).

Testing of ethylbenzene for carcinogenicity in animal bioassays has been deferred (NTP, 1985); therefore, ethylbenzene is classified an an IARC Group 3 chemical. Using the U.S. EPA, CAG rating system, ethylbenzene would be rated as a Group D chemical.

Ethyl_BENZENE

10. REFERENCES

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values, 4th ed. Cincinnati, OH. p. 176.

ACGIH (American Conference of Governmental Industrial Hygienists). 1985. TLVs: Threshold limit values for chemical substances and physical agents in the workroom environment with intended changes for 1985-1986. Cincinnati, OH. p. 18.

Amoore, J.E. and E. Hautala. 1983. Odor as an aid to chemical safety: Odor thresholds compared with threshold limit values and volatilities for 214 industrial chemicals in air and water dilution. J. Appl. Toxicol. 3(6): 272-290.

Anderson, R.A., I. Thomson and W.A. Harland. 1979. The importance of cyanide and organic nitriles in fire fatalities. Fire Mater. 3: 91-99.

Andersson, K., K. Fuxe, O.G. Nilsen, et al. 1981. Production of discrete changes in dopamine and noradrenaline levels and turnover in various parts of the rat brain following exposure to xylene, ortho-, meta-, and para-xylene, and ethylbenzene. Toxicol. Appl. Pharmacol. 60(3): 535-548.

Andrew, F.D., R.L. Buschbom, W.C. Cannon, R.A. Miller and B.D. Hardin. 1981. Teratologic assessment of ethylbenzene and 2-ethoxyethanol. NTIS PB83-208074, 108 p.

Angerer, J. and G. Lehnert. 1979. Occupational chronic exposure to organic solvents: VIII. Phenolic compounds: Metabolites of alkylbenzenes in man: Simultaneous exposure to ethylbenzene and xylenes. Int. Arch. Occup. Environ. Health. 43(2): 145-150.

Ball, G., B. Weiss and E.A. Boettner. 1970. Analysis of the volatile combustion products of poly(phenylene oxide) plastics. Am. Ind. Hyg. Assoc. J. 31(5): 572-578.

Banerjee, S., P.H. Howard, A.M. Rosenberg, A.E. Dombrowski, H. Sikka and D.L. Tullis. 1984. Development of general kinetic model for biodegradation and its application to chlorophenols and related compounds. Environ. Sci. Technol. 18: 416-422.

Bardodej, Z. and E. Bardodejova. 1970. Biotransformation of ethylbenzene, styrene and alpha-methylstyrene in man. Am. Ind. Hyg. Assoc. J. March-April: 206-209.

Barnes, R.D. and A.J. MacLeod. 1982. Analysis of the composition of the volatile malodor emissions from six animal rendering factories. Analyst (London): 711-715.

Battermann, G. and P. Werner. 1984. Removal of underground contamination by hydrocarbons through microbial degradation. GWF-Wasser/Abwasser. 125: 366-373. (Ger.)

Benville, P.E., Jr. and S. Korn. 1977. The acute toxicity of six monocyclic aromatic crude oil components to striped bass (Morone saxatilis) and bay shrimp (Crago franciscorum). Calif. Fish and Game. 63: 204-209.

Bertsch, W., E. Anderson and G. Holzer. 1975. Trace analysis of organic volatiles in water by gas chromatography-mass spectrometry with glass capillary columns. J. Chromatog. 112: 701-718.

Black, F. and L. High. 1977. Automotive hydrocarbon emission patterns in the measurement of nonmethane hydrocarbom emission rates. SAE (Tech. Pap), 770144. 16 p.

Bobra, A.M., W.Y. Shiu and D. Mackay. 1983. A predictive correlation for the acute toxicity of hydrocarbons and chlorinated hydrocarbons to the water flea (Daphnia magna). Chemosphere. 12: 1121-1129.

Bogan, R.H. and C.N. Sawyer. 1955. Biochemical degradation of synthetic detergents. II. Studies on the relation between chemical structure and biochemical oxidation. Sew. Ind. Wastes. 27: 917-928.

Bohon, R.L. and W.F. Claussen. 1951. The solubility of aromatic hydrocarbons in water. J. Am. Chem. Soc. 73: 1571-1578.

Bos, R., R. Guicherit and A. Hoogeveen. 1977. Distribution of some hydrocarbons in ambient air near Delft and the influence on the formation of secondary air pollutants. Sci. Total Environ. 7(3): 269-281.

Bouwer, E.J. and P.L. McCarty. 1984. Modeling of trace organics biotransformation in the subsurface. Ground Water. 22: 433-440.

Bouwer, E.J., P.L. McCarty, H. Bouwer and R.C. Rice. 1984. Organic contaminant behavior during rapid infiltration of secondary wastewater at the Phoenix 23rd Avenue Project. Water Res. 18: 463-472.

Bringmann, G. and R. Kuehn. 1976. Comparative results of the damaging effects of water pollutants against bacteria (Pseudomonas putida) and blue algae (Microcystis aeruginosa). Gas-Wasserfach, Wasser-Abwasser. 117(9): 410-413.

Bringmann, G. and R. Kuehn. 1977. Results of the damaging effect of water pollutants on Daphnia magna. Z. Wasser Abwasser Forsch. 10(5): 161-166.

Bringmann, G. and R. Kuehn. 1978. Testing of substances for their toxicity threshold: Model organisms Microcystis (Diplocystis) aeruginosa and Scenedesmus quadricauda. Mitt.-Int. Ver. Theor. Angew. Limnol. 21: 275-284.

Bringmann, G. and R. Kuehn. 1980. Comparison of the toxicity thresholds of water pollutants to bacteria, algae, and protozoa in the cell multiplication inhibition test. Water Res. 14: 231-241.

Bringmann, G. and R. Kuehn. 1982. Results of toxic action of water pollutants on Daphnia magna Straus tested by an improved standardized procedure. Z. Wasser Abwasser Forsch. 15(1): 1-6.

Brodzinsky, R. and H.B. Singh. 1982. Volatile organic chemicals in the atmosphere: An assessment of available data. Prepared under Contract No. 68-02-3452. Environmental Research Laboratory, ORD, U.S. EPA, Research Triangle Park, NC.

Brown, R.L. and S.P. Wasik. 1974. Method of measuring the solubilities of hydrocarbons in aqueous solutions. J. Res. Natl. Bur. Stand. Sec. A. 78: 453-560.

Buccafusco, R.J., S.J. Ellis and G.A. LeBlanc. 1981. Acute toxicity of priority pollutants to bluegill (Lepomis macrochirus). Bull Environ. Contam. Toxicol. 26: 446-452.

Burmaster, D.E. 1982. The new pollution-groundwater contamination. Environment. 24: 6-13, 33-36.

Carpenter, C.P., E.R. Kinkead, D.L. Geary, Jr., L.J. Sullivan and J.M. King. 1975. Petroleum hydrocarbon toxicity studies. V. Animal and human response to vapors of mixed xylenes. Toxicol. Appl. Pharmacol. 33(3): 543-558.

CEQ (Council of Environmental Quality). 1980. The 10 synthetic organic compounds most commonly detected in public water system wells in New York State, 1979. In: 11th Annual Report. CEQ, Washington, DC. 9[illegible]

Chao, J., C.T. Lin and T.H. Chung. 1983. Vapor pressure of coal chemicals. J. Phys. Chem. Ref. Data. 12: 1033-1063.

Chin, B.H., J.A. McKelvey, T.R. Tyler, L.J. Calisti, S.J. Kozbelt and L.J. Sullivan. 1980. Absorption, distribution, and excretion of ethylbenzene, ethylcyclohexane, and methylethylbenzene isomers in rats. Bull. Environ. Contam. Toxicol. 24(3): 477-483.

Chiou, C.T., P.E. Porter and D.W. Schemedding. 1983. Partition equilibria of nonionic organic compounds between soil, organic matter and water. Environ. Sci. Technol. 17: 227-231.

Clark, A.I., A.E. McIntyre, R. Perry and J.N. Lester. 1984a. Monitoring and assessment of ambient atmospheric concentrations of aromatic and halogenated hydrocarbons at urban, rural and motorway locations. Environ. Pollut. Ser. B. 7(2): 141-158.

Clark, A.I., A.E. McIntyre, J.N. Lester and R. Perry. 1984b. Ambient air measurements of aromatic and halogenated hydrocarbons at urban, rural and motorway locations. Sci. Total Environ. 39(3): 265-279.

Climie, I.J.G., D.H. Hutson and G. Stoydin. 1983. The metabolism of ethylbenzene hydroperoxide in the rat. Xenobiotica. 13(10): 611-618.

CMR (Chemical Marketing Reporter). 1983. Chemical Profile. Ethyl Benzene. Sept. 12, 1983.

Cocheo, V., M.L. Bellomo and G.G. Bombi. 1983. Rubber manufacture: Sampling and identification of volatile pollutants. Am. Ind. Hyg. Assoc. J. 44(7): 521-527.

Cole, R.H., R.E. Frederick, R.P. Healy and R.G. Rolan. 1984. Preliminary findings of the priority pollutant monitoring project of the nationwide urban runoff program. J. Water Pollut. Control Fed. 56: 898-908.

Coleman, W.E., J.W. Munch, R.P. Streicher, H.P. Ringhand and F.C. Kopfler. 1984. The identification and measurement of components in gasoline, kerosene and No. 2 fuel oil that partition into the aqueous phase after mixing. Arch. Environ. Contam. Toxicol. 13: 171-178.

Colenutt, B.A. and S. Thorburn. 1980. Gas chromatographic analysis of trace hydrocarbon pollutants in water samples. Int. J. Environ. Stud. 15: 25-32.

Cook, W.A. 1980. Industrial hygiene evaluation of thermal degradation products from PVC film in meat-wrapping operations. Am. Ind. Hyg. Assoc. J. 41(7): 508-512.

Cox, D.P. and C.D. Goldsmith. 1979. Microbial conversion of ethylbenzene to 1-phenethanol and acetophenone by Nocardia tartarsicans ATCC 31190. Appl. Environ. Microbiol. 38: 514-520.

Cupitt, L.T. 1980. Fate of Toxic and Hazardous Materials in the Air Environment. ESRL, ORD, U.S. EPA, Research Triangle Park, NC. EPA 600/3-80-084. NTIS PB80-221948.

Davis, J.B. and R.L. Raymond. 1961. Oxidation of alkyl-substituted cyclic hydrocarbons by a Nocardia during growth on N-alkanes. Appl. Microbiol. 9: 383-388.

Dean, B.J., T.M. Brooks, G. Hodson-Walker and D.H. Hutson. 1985. Genetic toxicology testing of 41 industrial chemicals. Mutat. Res. 153(1-2): 57-77.

Dilling, W.L. 1982. Atmospheric environment. Chapter 5. In: Environmental Risk Analysis for Chemicals, R.A. Conway, Ed. Van Nostrand Reinhold Co., New York, NY. p. 154-197.

Dilling, W.L., C.J. Bredeweg and N.B. Tefertiller. 1976. Organic photochemistry. XIII. Simulated atmospheric photodecomposition rates of methylene chloride, 1,1,1-trichloroethane, trichloroethylene, tetrachloroethylene, and other compounds. Environ. Sci. Technol. 10(4): 351-356.

Dimitriades, B., G.P. Sturm, T.C. Wesson and F.D. Sutterfield. 1975. Development and Utility of Reactivity Scales from Smog Chamber Data. RI 8023. U.S. Bureau of Mines, Pittsburgh, PA. p. 19.

Donner, M., J. Maeki-Paaxkanen, H. Norppa, M. Sorsa and H. Vainio. 1980. Genetic toxicology of xylenes. Mutat. Res. 74: 171-172.

Douglas, G.R., E.R. Nestmann, J.L. Betts, et al. 1980. Mutagenic activity in pulp mill effluents. Water Chlorination: Environ. Impact Health Eff. 3: 865-880.

Dowthy, B.J., D.R. Carlisle and J.L. Laseter. 1975. New Orleans drinking water sources tested by gas chromatography-mass spectrometry. Environ. Sci. Technol. 9: 762-765.

Durkin, P.R. 1985. Reference Values for Risk Assessment. Prepared by Syracuse Research Corporation, Syracuse, NY, under Contract No. 68-03-3228. ECAO, U.S. EPA, Cincinnati, OH.

Dwyer, F.G., P.J. Lewis and F.H. Schneider. 1976. Efficient nonpolling ethylbenzene process. Chem. Eng. 5: 90-91.

Ehrhardt, M. and G. Petrick. 1984. On the sensitized photooxidation of alkylbenzenes in seawater. Mar. Chem. 15: 47-58.

El Masry, A.M., J.N. Smith and R.T. Williams. 1956. Studies in detoxification: The metabolism of alkylbenzenes: n-Propylbenzene and n-butylbenzene with further observations on ethylbenzene. Biochem. J. 64: 50-56.

Elovaara, E., K. Engstrom, J. Nickels, A. Aitio and H. Vainio. 1985. Biochemical and morphological effects of long-term inhalation exposure of rats to ethylbenzene. Xenobiotica. 15(4): 299-308.

Engstrom, K.M. 1984. Metabolism of inhaled ethylbenzene in rats. Scand. J. Work Environ. Health. 10(2): 83-88.

Engstrom, J. and R. Bjurstrom. 1978. Exposure to xylene and ethylbenzene. II. Concentration in subcutaneous adipose tissue. Scand. J. Work Environ. Health. 4: 195-203.

Engstrom, K., E. Elovaara and A. Aitio. 1985. Metabolism of ethylbenzene in the rat during long-term intermittent inhalation exposure. Xenobiotica. 15(4): 281-286.

Esposito, G.G. 1968. Quantitative measure of photochemically reactive aromatic hydrocarbons in enamels and thinners. J. Paint Technol. 40: 214-221.

Farley, F.F. 1977. Photochemical Reactivity Classification of Hydrocarbons and Other Organic Compounds. Int. Conf. on Photochemical Oxidant Pollution and its Control, B. Dimitriades, Ed. U.S. EPA, Research Triangle Park, NC. EPA 600/3-77-001B. p. 713-727.

Feiler, H.D., A.S. Vernick and P.J. Storch. 1979. Fate of Priority Pollutants in POTWs. Proc. Natl. Conf. Munic. Sludge Manage. 8: 72-81.

Fel'dt, E.G. 1985. Evaluation of the mutagenic hazards of benzene and some of its derivatives. Gig. Sanit. 7: 21-23. [CA 103(13):99930h]

Ferrario, J.B., G.C. Lawler, I.R. Deleon and J.L. Laseter. 1985. Volatile organic pollutants in biota and sediments of Lake Pontchartrain. Bull. Environ. Contam. Toxicol. 34: 246-255.

Fielding, M, T.M. Gibson, H.A. James, K. McLoughlin and C.P. Steel. 1981. Organic micropollutants in drinking water. Water Res. Cent., Medmenham, England. 49 p.

Florin, I., L. Rutberg, M. Curvall and C.R. Enzell. 1980. Screening of tobacco smoke constituents for mutagenicity using the Ames' test. Toxicology. 15(3): 219-232.

Fraser, D.A. and S. Rappaport. 1976. Health aspects of the curing of synthetic rubbers. Environ. Health Perspect. 17: 45-53.

Gamberale, F., G. Annwall and M. Hultengren. 1978. Exposure to xylene and ethylbenzene. III. Effects on central nervous functions. Scand. J. Work, Environ, Health. 4(3): 204-211.

Gibson, D.T. 1977. Biodegradation of aromatic petroleum hydrocarbons. In: Fate and Effects of Petroleum Hydrocarbons in Marine Organism and Ecosystems, D.A. Wolfe, Ed. Pergamon Press, New York, NY. p. 36-46.

Gibson, D.T. 1978. Microbiol transformations of aromatic pollutants. In: Aquatic Pollutants. Trans. Biolog. Effects, O. Hutzinger et al., Ed. Pergamon Press, Oxford, England. p. 187-204.

Gibson, D.T. and W.K. Yeh. 1973. Microbial degradation of aromatic hydrocarbons. Microbial Degradation Oil Pollutants, Workshop. p. 33-38.

Gibson, D.T., J.R. Koch and R.E. Kallio. 1968. Oxidative degradation of aromatic hydrocarbons by microorganisms. I. Enzymic formation of catechol from benzene. Biochemistry. 7: 2653-2662.

Gibson, D.T., B. Gschwendt, W.K. Yeh and V.M. Kobal. 1973. Initial reactions in the oxidation of ethylbenzene. Biochemistry. 12: 1520-1528.

Glass, W.A. and C.S. Tuesday. 1970. Hydrocarbon reactivities in the atmospheric photooxidation of nitric oxide. Environ. Sci. Technol. 4(11): 916-924.

Goodley, P.C. and M. Gordon. 1976. Characterization of industrial organic compounds in water. Kentucky Acad. Sci. 37: 11-15.

Gossett, R.W., D.A. Brown and D.R. Young. 1983. Predicting the bioaccumulation of organic compounds in marine organisms using octanol/water partition coefficients. Mar. Pollut. Bull. 14(10): 387-392.

Graedel, T.E. 1978. Chemical Compounds in the Atmosphere. Academic Press, New York. p. 7.

Grob, K. 1973. Organic substances in potable water and in its precursor. Part I. Methods for this determination by gas-liquid chromatography. J. Chrom. 84: 255-273.

Grosjean, D. and K. Fung. 1984. Hydrocarbons and carbonyls in Los Angeles air. J. Air Pollut. Control Assoc. 34(5): 537-543.

Gschwend, P.M., D.C. Zafiriou, R.F.C. Mantoura, R.P. Schwarzenbach and R.B. Gagosian. 1982. Volatile organic compounds at a coastal site: 1. Seasonal variations. Environ. Sci. Technol. 16(1): 31-38.

Haemisegger, E.R., A.D. Jones and F.L. Reinhardt. 1985. EPA's experience with assessment of site-specific environmental problems: A review of IEMD's geographic study of Philadelphia. J. Air Pollut. Control Assoc. 35: 809-815.

Hagemann, J. and J. Angerer. 1979. Biological monitoring in occupational ethylbenzene loading. Krebsgefaehrdung Arbeitsplatz/Arbeitsmed. Kollog., Ber. Jahrestag. Dtsch. Ges. Arbeitsmed. 19: 421-425. (Ger.) (CA 94:196880v)

Hampton, C.V. W.R. Pierson, T.M. Harvey, W.S. Updergrove and R.S. Marano. 1982. Hydrocarbon gases emitted from vehicles on the road. 1. A qualitative gas chromatography-mass spectrometry survey. Environ. Sci. Technol. 16(5): 287-298.

Hampton, C.V., W.R. Pierson, T.M. Harvey and D. Schuetzle. 1983. Hydrocarbon gases emitted from vehicles on the road. 2. Determination of emission rates from diesel and spark-ignition vehicles. Environ. Sci. Technol. 17(12): 699-708.

Hansch, C. and A.J. Leo. 1985. MedChem Project. No. 26. Pomona College, Clarement, CA.

Hardin, B.D., G.P. Bond, M.R. Sikov, F.D. Andrew, R.P. Belliles and R.W. Niemeier. 1981. Testing of selected workplace chemicals for teratogenic potential. US-Finish Joint Symposium on Occupational Safety and Health and the 3rd Annual NIOSH Scientific Symposium, Cincinnati, OH and Detroit, MI, USA, Oct. 1980. Scand J. Work Environ. Health. 7(4): 66-75.

Harkov, R. 1982. Toxic air pollutants assessing their importance. Sci. Total Environ. 26(1): 67-86.

Harkov, R., B. Kebbekus, J.W. Bozzelli and P.J. Lioy. 1983. Measurement of selected volatile organic compounds at three locations in New Jersey during summer season. J. Air Pollut. Control Assoc. 33: 1177-1183.

Harkov, R., B. Kebbekus, J.W. Bozzelli, P.J. Lioy and J. Daisey. 1984. Comparison of selected volatile organic compounds during the summer and winter at urban sites in New Jersey. Sci. Total Environ. 38: 259-274.

Hawley, G.G. 1981. The Condensed Chemical Dictionary, 19th ed. Van Nostrand Reinhold Co., New York. p. 425.

Hawthorne, S.B. and R.E. Sievers. 1984. Emission of organic air pollutants from shale oil wastewaters. Environ. Sci. Technol. 18(6): 483-490.

Hawthorne, A.R., R.B. Gammage, C.S. Dudney, et al. 1983. Preliminary results of a forty-home indoor air pollutant monitoring study. In: Spec. Conf. Meas. Monit. Non-Criter. (Toxic) Contam. Air. p. 514-526.

Hendry, D.G. and R.A. Kenley. 1979. Atmospheric reaction products of organic compounds. Office of Chemical Control, OTS, U.S. EPA, Washington, DC. EPA 560/12-79-001.

Hendry, D.G., T. Mill, L. Piszkiewicz, J.A. Howard and H.K. Eigenmann. 1974. A critical review of H-atom transfer in the liquid phase: Chlorine atom, alkyl, trichloromethyl, alkoxy and alkylperoxy radicals. J. Phys. Chem. Ref. Data. 3: 944-978.

Herron, J.T. and R.E. Huie. 1973. Rate constant for the reactions of atomic oxygen (O3P) with organic compounds in the gas phase. J. Phys. Chem. Ref. Data. 2: 467-518.

Heuss, J.M. and W.A. Glasson. 1968. Hydrocarbon reactivity and eye irritation. Environ. Sci. Technol. 2(12): 1109-1116.

Higgins, I.J., D.J. Best and R.C. Hammond. 1980. New findings in methane-utilizing bacteria highlight their importance in the biosphere and their commercial potential. Nature. 286: 561-564.

Higgins, I.J., D.J. Best and D. Scott. 1981. Hydrocarbon oxidation by Methylosius trichosporium: Metabolic implications of the lack of specificity of methane monooxygenase. In: Microb. Growth C1 Compd. Proc. Int. Symp. 3: 11-20.

Higgins, C.E., W.H. Griest and G. Olerich. 1983. Application of Tenax trapping to analysis of gas-phase organic compounds in ultra-low tar cigaret smoke. J. Assoc. Off. Anal. Chem. 66(5): 1074-1083.

Holzer, G., J. Oro and W. Bertsch. 1976. Gas chromatographic-mass spectrometric evaluation of exhaled tobacco smoke. J. Chromatogr. 126: 771-785.

Horvath, R.S. 1972. Microbial co-metabolism and the degradation of organic compounds in nature. Bacteriol. Rev. 36: 146-55.

Hoshika, Y., Y. Nihei and G. Muto. 1981. Pattern display for characterization of trace amounts of odorants discharged from nine odor sources. Analyst (London). 106(1268): 1187-1202.

Hutchinson, T.C., J.A. Hellebust, D. Tam, D. Mackay, R.A. Mascarenhas and W.Y. Shiu. 1980. The correlation of the toxicity to algae of hydrocarbons and halogenated hydrocarbons with their physical-chemical properties. Environ. Sci. Res. 16: 577-586.

Jamison, V.W., R.L. Raymond and J.O. Hudson. 1970. Hydrocarbon cooxidation by Nocardia corallina strain V-49. Develop. Ind. Microbiol. 12: 99-105.

Jarke, F.H., A. Dravnieks and S.M. Gordon. 1981. Organic contaminants in indoor air and their relation to outdoor contaminants. ASHRAE Trans. 87(1): 153-166.

Juhnke, I. and D. Luedemann. 1978. Results of the study of 200 chemical compounds on acute fish toxicity using the Golden Orfe test. Z. Wasser Abwasser Forsch. 11(5): 161-164.

Junk, G.A. and C.S. Ford. 1980. A review of organic emissions from selected combustion processes. Chemosphere. 9(4): 187-230.

Kanatharano, P. and R.L. Grob. 1983. Gas chromatographic study of hydrocarbons absorbed on soils. J. Environ. Sci. Health, Part A. A18(1): 59-77. (CA 98:142346q)

Kanemitsu, H., M. Fukuda and K. Yano. 1980. Plasmid-borne biodegradation of toluene and ethylbenzene in a pseudomonad. J. Ferment. Technol. 58: 175-181.

Kappeler, T. and K. Whurmann. 1978a. Microbial degradation of the water soluble fraction of gas oil. I. Water Res. 12: 327-333.

Kappeler, T. and K. Wuhrmann. 1978b. Microbial degradation of the water-soluble fraction of gas oil: II. Bioassays with pure strains. Water Res. 12(5): 335-342.

Katzman, H. and W.F. Libby. 1975. Hydrocarbon emissions from jet engines operated at simulated high-altitude supersonic flight conditions. Atmos. Environ. 9(9): 839-842.

Kawamura, K. and I.R. Kaplan. 1983. Organic compounds in the rainwater of Los Angeles. Environ. Sci. Technol. 17: 497-501.

Kebbekus, B., A. Greenberg, L. Horgan, et al. 1983. Concentration of selected vapor and particulate-phase substances in the Lincoln and Holland Tunnels. J. Air Pollut. Control Assoc. 33(4): 328-330.

Keith, H., A.W. Garrison, F.R. Allen, et al. 1976. Identification of organic compound in drinking water from thirteen U.S. Cities. In: Identification and Analysis of Organic Pollutants in Water, L.H. Keith, Ed. Ann Arbor Press, Ann Arbor, MI. p. 329-373.

Kelley, R.D. 1985. Synthetic organic compound sampling survey of public water supplies. Iowa Dept. of Water, Air and Waste Management, Des Moines, IA. NTIS PB214427/XAB.

Kenaga, E.E. 1982. Predictability of chronic toxicity from acute toxicity of chemicals in fish and aquatic invertebrates. Environ. Toxicol. Chem. 1(4): 347-358.

Kimmel, C.A. and J.G. Wilson. 1973. Skeletal deviations in rats: Malformations or variations. Teratology. 8: 309-315.

Kleopfer, R.D. and B.J. Fairless. 1972. Characterization of organic components in a municipal water supply. Environ. Sci. Technol. 6(12): 1036-1037.

Knaggs, E.A., M.L. Nussbaum and A. Shultz. 1983. Sufonation and sulfation. In: Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 22. John Wiley and Sons, New York. p. 7.

Kopczynski, S.L. 1964. Photooxidation of alkylbenzene-nitrogen dioxide mixtures in air. Int. J. Air Water Pollut. 8: 107-120.

Kopczynski, S.L., W.A. Lonneman, F.D. Sutterfield and P.E. Darley. 1972. Photochemistry of atmospheric samples in Los Angeles. Environ. Sci. Technol. 6: 342.

Kopfler, F.C., R.G. Melton, J.L. Mullaney and R.G. Tardiff. 1975. In: Advances in Environmental, Science and Technology, Vol. 8. Fate of Pollutants in the Air and Water Environments. Part 2. Chemical and Biological Fate of Pollutants in the Environment. Symp. 165th Meet. NACS, Environ. Chem. Div., Philadelphia, PA. April 1975. 442 p.

Kover, F.D. 1980a. Status Report to J.J. Merenda, Director Assessment Division, U.S. EPA, Washington, DC. Sept. 16, 1980.

Kover, F.D. 1980b. Status Report to J.J. Merenda, Director Assessment Division, U.S. EPA, Washington, DC. Jan. 22, 1980.

Kozloski, R.P. and B.L. Sawhney. 1982. Multiple purge technique for determining organic pollutants in ground water. Bull. Environ. Contam. Toxicol. 29(1): 1-6.

Laity, J.L., I.G. Burstain and B.R. Appel. 1973. Photochemical smog and the atmospheric reactions of solvents. Solvent Theory and Practice, Advan. Chem. Ser. 124: 95-112.

LeGore, R.S. 1974. The effect of Alaskan crude oil and selected hydrocarbon compounds on embryonic development of the Pacific oyster, Crassostrea gigas. Doctoral Thesis, Univ. Washington. (Cited in U.S. EPA, 1980)

Levine, S.P., R.J. Costello, C.L. Ceraci and K.A. Conlin. 1985. Air monitoring at the drum bulking process of a hazardous waste remedial action site. Am. Ind. Hyg. Assoc. J. 46(4): 192-196.

Levy, A. 1973. The photochemical smog reactivity of organic solvents. Solvent theory and practices. American Chemical Society, Washington, DC. Adv. Chem. Ser. 124: 70-94.

Lewis, P.J., C. Hagopian and P. Koch. 1983. Styrene. In: Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 21. John Wiley and Sons, New York. p. 772-779.

Lloyd, A.C., K.R. Darnall, A.M. Winer and J.N. Pitts, Jr. 1976. Relative rate constants for reaction of the hydroxyl radical with series of alkanes, alkanes and aromatic hydrocarbons. J. Phys. Chem. 80: 789-794.

Ludzack, F.J. and M.B. Ettinger. 1963. Biodegradability of organic chemicals isolated from rivers. Proc. of the 18th Ind. Waste Conf. Purdue Univ., Eng. Bull., Ext. Series. 18: 278-282.

Lyman, W.J., W.F. Reehe and D.H. Rosenblatt. 1982. Handbook of Chemical Property Estimation Methods. Environmental Behavior of Organic Compounds. McGraw-Hill Book Co., New York. 960 p.

Mabey, W.R., J.H. Smith, R.T. Podoll, et al. 1981. Aquatic Fate Process Data for Organic Priority Pollutants. Monitoring and Data Support Div., Office of Water Regulations and Standards, Washington, DC. EPA 440/4-81-014.

MacFarlane, A.C. 1976. Ethylbenzene process proves out oils. Gas. J. 74: 99-102.

Mackay, D. and W.Y. Shiu. 1981. A critical review of Henry's Law Constant for chemicals of environmental interest. J. Phys. Chem. Ref. Data. 19: 1175-1199.

Malaney, G.W. 1960. Oxidative abilities of aniline-acclimated activated sludge. J. Water Pollut. Control Fed. 32: 1300.

Malaney, G.W. and R.E. McKinney. 1966. Oxidative abilities of benzene-acclimated activated sludge. Water Sewage Works. 113: 302-309.

Malle, K.G. 1984. The importance of the 129 substances on the list of priority pollutants for water protection. Z. Wasser-Abwasser Forsch. 17: 75-81. (Ger.)

Marion, C.V. and G.W. Malaney. 1964. Ability of activated sludge micro-organisms to oxidize aromatic organic compounds. Proc. 18th Ind. Waste Conf. Eng. Bull. Purdue Univ., Eng. Ext. Ser. p. 297-308.

McAuliffe, C. 1963. Solubility in water of C1-C9 hydrocarbons. Nature. 200: 1092-1093.

McAuliffe, C. 1966. Solubility in water of paraffin, cycloparaffin, olefin, acetylene, cycloolefin and aromatic hydrocarbon. J. Phys. Chem. 70: 1267-1275.

Mill, T., W.R. Malay, D.C. Bomberger, et al. 1982. Laboratory Protocols for Evaluating the Fate or Organic Chemicals in Air and Water. Environmental Research Laboratory, ORD, U.S. EPA, Athens, GA. EPA 600/3-82-022.

Morrow, J.E., R.L. Gritz and M.P. Kirton. 1975. Effects of some components of crude oil on young coho salmon. Copeia. 1: 326-331.

Mottram, D.S., S.E. Croft and R.L.S. Patterson. 1984. Volatile components of cured and uncured pork: The role of nitrite and the formation of nitrogen compounds. J. Sci. Food Agric. 35(2): 233-239.

Myers, V.B. 1983. Remedial activities at the Miami Drum site, Florida. In: Natl. Conf. Manag. Uncont. Hazard. Waste Sites. p. 354-357.

NAS (National Academy of Sciences). 1980. The Alkyl Benzenes. National Academy Press, Washington, DC.

NCI (National Cancer Institute). 1985. Monograph on Human Exposure to Chemicals in the Workplace: Styrene. Prepared by Syracuse Research Corporation, Syracuse, NY., under Contract No. 1-CP-26002-03. Division of Cancer Etiology, NCI, Bethesda, MD.

Nelson, P.F. and S.M. Quigley. 1982. Non-methane hydrocarbons in the atmosphere of Sydney, Australia. Environ. Sci. Technol. 16: 650-655.

Nestmann, E.R. and E.G.H. Lee. 1983. Mutagenicity of constituents of pulp and paper mill effluent in growing cells of Saccharomyces cerevisiae. Mutat. Res. 119(3-4): 273-280.

Nestmann, E.R., E.G.H. Lee, T.I. Matula, G.R. Douglas and J.C. Mueller. 1980. Mutagenicity of constituents identified in pulp and paper mill effluents using the Salmonella/mammalian-microsome assay. Mutat. Res. 79: 203-212.

Norppa, H. and H. Vainio. 1983. Induction of sister-chromatid exchanges by styrene analogs in cultured human lymphocytes. Mutat. Res. 116(3-4): 379-387.

NTP (National Toxicology Program). 1985. Management Status Report. 11/4/85.

Nunes, P. and P.E. Benville. 1979. Uptake and depuration of petroleum hydrocarbons in the Manila clam, Tapes semidecussata reeve. Bull. Environ. Contam. Toxicol. 21(6): 719-726.

Oduntan, D. and O. Odeyemi. 1984. Relative half-lives of chloromil and dichlone in different tropical miheux. Environ. Poll. (Ser. A). 36: 327-335.

Ogata, M., K. Fujisawa, Y. Ogino and E. Mano. 1984. Partition coefficients as a measure of bioconcentration potential of crude oil compounds in fish and shellfish. Bull. Environ. Contam. Toxicol. 33(5): 561-567.

OSHA (Occupational Safety and Health Administration). 1985. Permissible Exposure Limits. Code of Federal Regulations. 29: 1910.1000.

Otson, R., D.T. Williams and D.C. Biggs. 1982. Relationships between raw water quality treatment and occurrence of organics in Canadian potable water. Bull. Environ. Contam. Toxicol. 28(4): 396-403.

Pankow, J.F., L.M. Isabelle and W.E. Asher. 1984. Trace organic compounds in rain. I. Sampler design and analysis by adsorption/thermal desorption (ATD). Environ. Sci. Technol. 18: 310-318.

Patterson, J.W. and P.S. Kodukala. 1981. Biodegradation of hazardous organic pollutants. Chem. Eng. Prog. 77: 48-55.

Pellizzari, E.D., T.D. Hartwell, B.S.H, Harris III, R.D. Waddell, D.A. Whitaker and M.D. Erickson. 1982. Purgeable organic compounds in mother's milk. Bull. Environ. Contam. Toxicol. 28(3): 322-328.

Perry, J.J. 1979. Microbial co-oxidations involving hydrocarbons. Microbiol. Rev. 43: 59-72.

Perry, R. and J.D. Twibell. 1973. A time-based elution technique for the estimation of specific hydrocarbons in air. Atmos. Environ. 7: 929-937.

Petersson, G. 1982. Ambient hydrocarbons from motor-car assembly plants in Scandinavia. Environ. Pollut. Ser. B. 4(3): 207-217.

Pickering, Q.H. and C. Henderson. 1966. Acute toxicity of some important petrochemical to fish. J. Water Pollut. Control Fed. 38: 1419-1429.

Polak, J., and B.C.Y. Lu. 1973. Mutual solubilities of hydrocarbons and water at 0 and 25°C. Can. J. Chem. 51: 4018-4033.

Potenta, E.J. and M.F. Saunders. 1983. Indoor air quality investigation of chronic health problems in a Manhattan office space. In: Proc. 76th Ann. Meet. Air Pollut. Control Assoc. 4: 83-64.6

Potera, F.T. 1975. The effects of benzene, toluene, and ethylbenzene on several important members of the estuarine ecosystem. Ph.D dissertation, Lehigh Univ. (Cited in U.S. EPA, 1980)

Rappaport, S.M. and D.A. Fraser. 1977. Air sampling and analysis in a rubber vulcanization area. J. Am. Ind. Hyg. Assoc. 38(5): 205-210.

Ravishankara, A.R., S. Wagner, S. Fischer, et al. 1978. A kinetics study of the reactions of hydroxyl with several aromatic and olefinic compounds. Int. J. Chem. Kinet. 10: 783-804.

Rawlings, G.D. and M. Samfield. 1979. Toxicity of secondary effluents from textile plants. ORD, U.S. EPA, Washington, DC. EPA 600/7-78-168.

Reinhard, M., N.L. Goodman and J.F. Barker. 1984. Occurrence and distribution of organic chemicals in two landfill leachate flumes. Environ. Sci. Technol. 18: 953-961.

Ribbons, D.W. and R.W. Eaton. 1982. Chemical transformations of aromatic hydrocarbons that support the growth of microorganisms. In: Biodegradation and Detoxification of Environmental Pollutants, A.M. Chakrabarty, Ed. CRC Press, Inc. p. 59-84.

Rosen, A.A., R.J. Skeel and M.B. Ettinger. 1963. Relationship of river water odor to specific organic contaminants. J. Water Pollut. Control Fed. 35: 777-782.

Sabel, G.V. and T.P. Clark. 1984. Volatile organic compounds as indicators of municipal solid waste leachate contamination. Waste Manag. Res. 2: 119-130.

Sabljic, A. 1984. Predictions of the Nature and strength of soil sorption of organic pollutants by molecular topology. J. Agric. Food Chem. 32: 243-246.

Sadtler. 1966. Sadtler Standard Spectra. Sadtler Research Lab, Philadelphia, PA.

Sanemasa, I., M. Araki, T. Deguchi and H. Nagai. 1981. Solubilities of benzene and the alkylbenzenes in water. Methol for obtaining aqueous solutions saturated with vapors in equilibrium with organic liquids. Chem. Lett. 2: 225-228.

Sauer, T.C., Jr., W.M. Sackett and L.M. Jeffrey. 1978. Volatile liquid hydrocarbons in the surface coastal waters of the Gulf of Mexico. Mar. Chem. 7: 1-16.

Schoenberg, M.R., J.W. Blieszner and C.G. Papadopoulos. 1982. Propylene. In: Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 19. John Wiley and Sons, New York. p. 240.

Schwarz, F.P. and J. Miller. 1980. Determination of the aqueous solubilities of organic liquids at 10, 20 and 30°C by election chromatography. Anal. Chem. 52: 2162-2164.

Seila, R.L., R.R. Arnsts and J.W. Buchanan. 1984. Atmospheric volatile hydrocarbon composition at five remote sites in northeastern North Carolina. U.S. EPA, Research Triangle Park, NC. EPA-600-D-84-092. NTIS PB 84-092.

Sexton, K. and H. Westberg. 1980a. Ambient hydrocarbon and ozone measurements downwind of a large automotive painting plant. Environ. Sci. Technol. 14(3): 329-323.

Sexton, K. and H. Westberg. 1980b. Elevated ozone concentrations measured downwind of the Chicago-Gary urban complex. J. Air Pollut. Control Assoc. 30(8): 911-914.

Shen, T.T. 1982. Air quality assessment for land disposal of industrial wastes. Environ. Management. 6: 297-305.

Singh, H.B., L.J. Salas, R. Stiles and H. Shigeishi. 1981. Atmospheric measurements of selected hazardous organic chemicals. U.S. EPA, Research Triangle Park, NC. EPA-600/93-81-032.

Smyth, H.F., C.P. Carpenter, C.S. Weil, U.C. Pozzani and J.A. Stiegel. 1962. Range-finding toxicity data: List VI. Ind. Hyg. J. p. 95-107.

Snider, E.H. and F.S. Manning. 1982. A survey of pollutant emission levels in wastewaters and residuals from the petroleum refining industry. Environ. Int. 7(4): 237-238.

SRI (Stanford Research Institute). 1985. 1985 Directory of Chemical Producers, United States of America. SRI International, Menlo Park, CA. p. 566.

Staples, C.A., A. Werner and T. Hoogeveem. 1985. Assessment of priority pollutant concentrations in the United States using STORET data base. Environ. Toxicol. Chem. 4: 131-142.

Stavinoha, L.L. and F.M. Newman. 1972. The isolation and determination of aromatics in gasoline by gas chromatography. Chrom. Sci. 10: 583-589.

Stuermer, D.H., D.J. Ng and C.J. Morris. 1982. Organic contaminants in groundwater near an underground coal gasification site in northeastern Wyoming. Environ. Sci. Technol. 16: 582-587.

Suffet, I.H., L. Brenner and J.V. Radziul. 1976. GC/MS identification of trace organic compounds in Philadelphia waters. In: Identification and Analysis of Organic Pollutants in Water, L.H. Keith, Ed. Ann Arbor Science Publ., Ann Arbor, MI. p. 375-397.

Sutton, C. and J.A. Calder. 1975. Solubility of alkylbenzenes in distilled water and sea water at 25°C. J. Chem. Eng. Data. 20: 320-322.

Tabak, H.H., S.A. Quave, C.I. Mashni and E.F. Barth. 1981. Biodegradability studies with organic priority pollutant compounds. J. Water Pollut. Control Fed. 53: 1503-1518.

Tatrai, E., T. Balogh, G. Barcza and G. Ungvary. 1982. Embryotoxic effect of ethylbenzene. Egeszsegtudomany. 26(3): 297-303. (CA 100:46781s)

Tester, D.J. and R.J. Harker. 1981. Groundwater pollution investigations in the Great Ouse Basin. Water Pollut. Control. 80: 614-631.

Tewari, Y.B., M.M. Miller, S.P. Wasik and D.E. Martine. 1982. Aqueous solubility and octanol/water partition coefficient of organic compounds at 25°C. J. Chem. Eng. Data. 27: 451-454.

Tham, R., I. Bunnfors, B. Eriksson, B. Larsby, S. Lindgren and L.M. Odkvist. 1984. Vestibulo-ocular disturbances in rats exposed to organic solvents. Acta Pharmacol. Toxicol. 54(1): 58-63.

Thom, N.S. and A.R. Agg. 1975. The breakdown of synthetic organic compounds in biological processes. Proc. R. Soc. Lond. B. 189: 347-357.

Toftgaard, R. and O.G. Nilsen. 1981. Induction of cytochrome P-450 in rat liver after inhalation of aromatic organic solvents. Ind. Environ. Xenobiotics, Proc. Int. Conf. Meeting Date 1980. p. 307-317.

Toftgaard, R. and O.G. Nilsen. 1982. Effects of xylene and xylene isomers on cytochrome P-450 and *in vitro* enzymic activities in rat liver, kidney and lung. Toxicology. 23(2-3): 197-212.

U.S. EPA. 1977. Computer Print-out of Nonconfidential Production Data from TSCA Inventory. OPTS, CID, U.S. EPA, Washington, DC.

U.S. EPA. 1978. In-depth Studies on Health and Environmental Impacts of Selected Water Pollutants. Contract No. 68-01-4646.

U.S. EPA. 1980. Ambient Water Quality Criteria for Ethylbenzene. Office of Water, Regulations and Standards, Criteria and Standard Div., U.S. EPA, Washington, DC. EPA 440/5-80-048. NTIS PB 81-117590.

U.S. EPA. 1983. Reportable Quantity Document for Ethylbenzene. Prepared for Office of Solid Waste and Emergency Response by ECAO, Cincinnati, OH.

U.S. EPA. 1985a. Drinking Water Criteria Document for Ethylbenzene. Prepared for the Office of Drinking Water by the ECAO, U.S. EPA, Cincinnati, OH. NTIS PB86-117835/AS.

U.S. EPA. 1985b. Summary of Acceptable Daily Intakes (ADIs) for Oral Exposure. Prepared by ECAO, Cincinnati, OH.

USITC (U.S. International Trade Commission). 1978. Synthetic Organic Chemicals United States Production and Sales, 1977. USITC Publ. 920. USITC, Washington, DC. p. 27.

USITC (U.S. International Trade Commission). 1985. Synthetic Organic Chemicals United States Production and Sales, 1984. USITC Publ. 1745. USITC, Washington, DC. p. 25, 38.

Van Der Linden, A.C. 1978. Degradation of oil in the marine environment. Dev. Biodegrad. Hydrocarbons. 1: 165-200.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., New York, NY. p. 628-630.

Vol'Fovskaya, R.N. and I.D. Makulova. 1971. Capacity for work in cases of chronic industrial intoxications, with symptoms of neurocirculatory dystonia. Nauchn Tr Leninger. Inst. Usoversh. Vrachei. Im. S. M. Kirova. 98: 151-155. (Taken from HEEP: Biol. Abs./73/10365)

Wakeham, S.G., A.C. Davis and J.L. Karas. 1983. Mesocosm experiments to determine the fate and persistence of volatile organic compounds in coastal seawater. Environ. Sci. Technol. 17: 611-617.

Wallace, L.A., E.D. Pellizzari, T.D. Hartwell, C. Sparacino and H. Zelon. 1983. Personal exposure to volatile organics and other compounds indoors and outdoors - the TEAM study. In: Proc 76th Ann. Meet. Air Pollut. Control Assoc. 1: 83-9.12.

Wasik, S.P., M.M. Miller, Y.B. Tewari, et al. 1983. Determination of the vapor pressure, aqueous solubility, and octanol/water partition coefficient of hydrophobic substances by coupled generator column/liquid chromatographic methods. Residue Rev. 85: 29-42.

Westberg, H., K. Sexton and D. Flyckt. 1981. Hydrocarbon production and photochemical ozone formation in forest burn plumes. J. Air Pollut. Control Assoc. 31(6): 661-664.

Westrick, J.J., J.W. Mello and R.F. Thomas. 1984. The groundwater supply survey. J. Am. Water Works Assoc. 76: 52-59.

Whitehead, L.W., G.L. Ball, L.J. Fine and G.D. Langolf. 1984. Solvent vapor exposures in booth spray painting and spray gluing and associated operations. Am. Ind. Hyg. Assoc. J. 45(11): 767-762.

Wolf, M.A., V.K. Rowe, D.D. McCollister, R.L. Hollingsworth and F. Oyen. 1956. Toxicological studies of certain alkylated benzenes and benzene. Arch. Ind. Health. 14: 387-398.

Yamasaki, Y. 1984. Determination of urinary metabolites of ethylbenzene in men by high performance liquid chromatography. Okayama Igakkai Zasshi. 96(5-6): 531-535. (CA 102:107702g)

Yanagihara, S., I. Shimada, E. Shinoyama, F. Chisaka and K. Saito. 1977. Photochemical reactivities of hydrocarbons. In: Proc. Int. Clean Air Congr., 4th 472-7, N. Kasuga, T. Susuki, T. Yamada, Ed. Jap. Union Air Pollut. Prev. Assoc., Tokyo, Japan.

Young, D.R., R.W. Gossett, R.B. Baird, D.A. Brown, P.A. Taylor and M.J. Miille. 1983. Wastewater inputs and marine bioaccumulation of priority pollutant organics off Southern California. Water Chlorination: Environ. Impact Health Eff. 4(2): 871-884.

Zoeteman, B.C.J., E. DeGreef and F.J.J. Brinkman. 1981. Persistence of organic contaminants in ground water, lessons from soil pollution incidents in The Netherlands. Sci. Total Environ. 21: 187-202.

APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

CASR online (U.S. EPA Chemical Activities Status Report)
CAS onliine STN Internation
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in November, 1985. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values, 4th ed. (Includes Supplemental Documentation, 1981, 1982, 1983). Cincinnati, OH. 486 p.

ACGIH (American Conference of Governmental Industrial Hygienists). 1985. TLVs: Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1985-1986. Cincinnati, OH. 114 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2A. John Wiley and Sons, NY. p. 2878.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2B. John Wiley and Sons, NY. p. 2879-3816.

Clayton, G.D. and F.E. Clayton, Ed. 1982. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2C. John Wiley and Sons, NY. p. 3817-5112.

Grayson, M. and D. Eckroth, Ed. 1978-1983. Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed. John Wiley and Sons, NY. 23 Volumes.

Hamilton, A. and H.L. Hardy. 1974. Industrial Toxicology, 3rd ed. Publishing Sciences Group, Inc., MA. 575 p.

IARC (International Agency for Research on Cancer). IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. WHO, IARC, Lyons, France.

ITII (International Technical Information Institute). 1982. Toxic and Hazardous Industrial Chemicals Safety Manual for Handling and Disposal with Toxicity and Hazard Data. ITII, Tokyo, Japan. 700 p.

NTP (National Toxicology Program). 1984. Toxicology Research and Testing Program. Chemicals on Standard Protocol. Management Status.

Ouellette, R.P. and J.A. King. 1977. Chemical Week Pesticide Register. McGraw-Hill Book Co., NY.

Sax, N.I. 1979. Dangerous Properties of Industrial Materials, 5th ed. Van Nostrand Reinhold Co., NY.

SRI (Stanford Research Institute). 1984. Directory of Chemical Producers. Menlo Park, CA.

U.S. EPA. 1985. Status Report on Rebuttable Presumption Against Registration (RPAR) or Special Review Process. Registration Standards and the Data Call in Programs. Office of Pesticide Programs, Washington, DC.

U.S. EPA. 1985. CSB Existing Chemical Assessment Tracking System. Name and CAS Number Ordered Indexes. Office of Toxic Substances, Washington, DC.

USITC (U.S. International Trade Commission). 1983. Synthetic Organic Chemicals. U.S. Production and Sales, 1982, USITC Publ. 1422. Washington, DC.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., NY.

Worthing, C.R. and S.B. Walker, Ed. 1983. The Pesticide Manual. British Crop Protection Council. 695 p.

Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book. Volume 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under Contract No. 68-01-0007. Washington, DC.

Johnson, W.W. and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory. 1965-1978. USDI, Fish and Wildlife Serv. Res. Publ. 137, Washington, DC.

McKee, J.E. and H.W. Wolf. 1963. Water Quality Criteria, 2nd ed. Prepared for the Resources Agency of California, State Water Quality Control Board. Publ. No. 3-A.

Pimental, D. 1971. Ecological Effects of Pesticides on Non-Target Species. Prepared for the U.S. EPA, Washington, DC. PB-269605.

Schneider, B.A. 1979. Toxicology Handbook. Mammalian and Aquatic Data. Book 1: Toxicology Data. Office of Pesticide Programs, U.S. EPA, Washington, DC. EPA 540/9-79-003. NTIS PB 80-196876.